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LONG-TERM ANTICOAGULANT THERAPY IN MYOCARDIAL INFARCTION*

A PRELIMINARY REPORT

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Anticoagulant therapy during the acute phase of myocardial infarction, has been shown to reduce the mortality of the disease. This has been explained mainly by the lowering of the incidence of the thrombo-embolic complications which result from thrombus formation occurring on the endocardium and in the veins of the extremities.[1, 2] The prevention of the propagation of an existing thrombus in a coronary vessel, thereby limiting the extent of the myocardial infarct, may possibly be an additional factor in lowering the mortality. That this may be true is supported by the observation that, in dogs, the thrombus formation and consequent myocardial infarction, resulting from the intra-arterial injection of sclerosing agents, was inhibited by the simultaneous administration of heparin.[3] However, following coronary artery ligation, heparin and dicoumarol (bishydroxycoumarin) failed to prevent arterial thrombosis and, moreover, neither a reduction in the size of the resulting infarct nor a greater or more rapid development of collateral vessels was observed.[4-6]

It has been shown in dogs that both dicoumarol and the sodium salts of heparin, acting directly on the vessel wall, induce vasodilatation and thereby increase the coronary blood flow. That dicoumarol may act in a similar manner on the coronary vessels in man is supported by the observation that, following myocardial infarction, the incidence of severe congestive failure and other manifestations of coronary insufficiency is lessened when the patients were treated with dicoumarol.[7]

It has also been observed,[8] though not confirmed,[9] that patients with angina pectoris derived benefit from the intravenous administration of heparin in doses of 50 to 100 mg. at weekly or biweekly intervals. Because of the low dosage and the infrequency of injections it was considered unlikely that the beneficial effects were due to the anticoagulant or vasodilator actions of heparin, but rather that the clinical improvement was dependent upon the action of heparin in altering the particular lipoprotein pattern thought to be concerned in the pathogenesis of atheroma.

Since it is generally accepted that atheroma is the underlying pathological process which leads to both acute and progressive coronary artery occlusion, it is of interest that when an intra-arterial mural thrombus, experimentally produced, has undergone organization and endothelialization, it presents as a lesion histologically indistinguishable from that of atheroma, even to the extent of exhibiting the characteristic fatty changes, calcification and ulceration.[10-12] Based on the concept that atheroma may result from intra-arterial mural thrombus formation rather than from a lesion arising subendothelially in the vessel wall, the long-continued administration of anticoagulants would appear to be rational therapy for the prevention of progressive coronary artery disease.

Reports have appeared concerning the use of long-term anticoagulant therapy in coronary thrombosis with myocardial infarction. In a patient with myocardial infarction and multiple recurrent thrombo-embolic phenomena, dicoumarol, administered over a period of 51 months, prevented further such episodes.[13] A series of 78 patients with myocardial infarction, of whom approximately one half had experienced previous attacks of coronary thrombosis, were observed for 3 months to 5 years while receiving continuous dicoumarol therapy.[14] The prothrombin time was estimated at weekly or fortnightly intervals and the daily maintainance dose of dicoumarol adjusted accordingly to maintain a prothrombin time of twice the normal. Haemorrhage occurred in approximately one third of the patients, necessitating cessation of therapy in 3. Of 12 patients who died while receiving anticoagulants, death was due to a recurrent attack of coronary thrombosis in 4, to congestive cardiac failure, acute coronary insufficiency or sudden arrest of cardiac activity

* Read at the Johannesburg Congress of the South African Medical Association in September 1952.
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in 6 and to the toxic effects of dicoumarol in 1 patient. Four of the surviving patients experienced episodes of acute coronary insufficiency with possible subendocardial infarction, but in none did recurrent attacks of frank coronary thrombosis occur. While no conclusions were drawn with regard to the efficacy of therapy, it was considered that the regime was feasible provided accurate prothrombin control was maintained. However, further extensive well-controlled clinical trials were deemed necessary in order to verify the possible value of this form of therapy.

The purpose of this paper is to compare the prognosis of myocardial infarction in a group of patients receiving long-term anticoagulant therapy with that observed in a group of patients not so treated. The results in the 2 groups are evaluated in respect of the mortality and the incidence of attacks of recurrent acute coronary thrombosis, acute coronary insufficiency, angina pectoris and cardiac failure.

MATERIAL AND METHODS

Eighty-five patients, all of whom received both heparin and dicoumarol, were observed during the acute phase of myocardial infarction and of these 11 died. Of the 74 patients surviving, continuous anticoagulant therapy was instituted in 37,* but of these, 8 discontinued treatment after periods varying from 1 to 9 months. Thus, 29 patients have continued with treatment to date. Of the 37 patients in whom continuous therapy was not instituted, 22 have been traced and serve as a control group.

The age of the patients in the control group ranged from 32 to 79 years with an average of 55.5 years and in the treated group from 40 to 77 years with an average of 52.7 years. There were 17 males and 5 females in the control group and 26 males and 3 females in the treated group. A previous history of coronary artery disease was elicited in 3 of the patients in the control group and in 5 of the treated. The numbers of cases and the periods of observation in both groups are shown in Table 1. The average period of observation of the patients in the control group was 31 months and in the treated group 27 months.

TABLE 1: THE NUMBERS OF PATIENTS, TREATED AND UNTREATED, OBSERVED FOR VARYING PERIODS

Periods of Observation (Months)	*Numbers of Cases*	
	Control Group	*Treated Group*
Less than 12	3	6
12 to 23	4	6
24 to 35	10	11
36 to 47	4	3
48 and over	1	3
Total	22	29

The anticoagulant used was dicoumarol in all except 2 patients who received Tromexan, one throughout the course and the other for a short period in place of dicoumarol. The prothrombin time, recorded as the index, was determined at weekly or fortnightly intervals and the dose of anticoagulant adjusted accordingly, to maintain as close as possible a level of prothrombin corresponding to an index of 50%.

* Fifteen of these were under the private care of one of us (M. M. S.).

RESULTS

The numbers of patients in each group who had subsequent attacks of acute coronary insufficiency, angina pectoris, cardiac failure or recurrent episodes of acute coronary thrombosis is shown in Table 2. Some of the patients with angina pectoris developed cardiac failure and/or recurrent attacks of acute coronary thrombosis.

TABLE 2

The numbers of patients, treated and untreated, who, subsequent to recovery from the acute phase of myocardial infarction developed clinical manifestations of progressive coronary artery disease and the numbers of patients with each particular complication who have since died or are surviving.

	Numbers of Cases					
	Control Group (22 Cases)			*Treated Group (29 Cases)*		
	Total	*Dead*	*Living*	*Total*	*Dead*	*Living*
Angina Pectoris and Acute Coronary Insufficiency	9	5	4	14	0	14
Cardiac Failure	9	5	4	4	1	3
Recurrent Acute Coronary Thrombosis	6	6	0	4	0	4

These patients are represented in the table not only amongst the patients with angina pectoris but also amongst those with cardiac failure and/or amongst those with recurrent coronary thrombosis. Similarly, some of the patients with recurrent coronary thrombosis developed cardiac failure and are represented in the table both amongst those with recurrent coronary thrombosis and amongst those with cardiac failure.

Angina Pectoris and Acute Coronary Insufficiency. In the control group 7 patients had angina pectoris subsequent to the acute attack of myocardial infarction. Two of these as well as 2 further patients had episodes of acute coronary insufficiency. Five of these patients have subsequently died, 1 suddenly, 1 as a result of recurrent coronary thrombosis and 3 of congestive cardiac failure. Of the surviving 4 patients, angina is severe in 2 and of a mild nature in the remaining 2 patients.

In the treated group 12 patients had subsequent angina pectoris. One of these as well as 2 further patients had episodes of acute coronary insufficiency. None of these patients has since died or developed cardiac failure but in 2 recurrent attacks of coronary thrombosis have occurred. Angina has persisted in a severe form in 3 patients and to a milder degree in 4, while in 5 patients the attacks have either diminished in severity or disappeared.

Cardiac Failure. In the control group cardiac failure occurred in 9 patients, in 5 of whom it was fatal.

In the treated group cardiac failure developed in 4 patients, in 1 of whom it was fatal.

Recurrent Acute Coronary Thrombosis. In the control group recurrent coronary thrombosis occurred in 6

patients. One of these had 2 attacks. It was the cause of death in 4 and precipitated fatal congestive cardiac failure in 2 patients.

In the treated group, 4 patients had each a single subsequent attack of acute coronary thrombosis. In none of these did cardiac failure or death ensue.

Table 3 indicates the numbers of patients in each group who ultimately died of the effects of ischaemic heart disease and the immediate cause of death.

TABLE 3: THE NUMBERS OF DEATHS AND THE IMMEDIATE CAUSES OF DEATH IN THE TREATED AND UNTREATED PATIENTS

	Numbers of Cases	
	Control Group (22 Cases)	*Treated Group (29 Cases)*
Cardiac Failure	5	1
Acute Coronary Thrombosis	4	0
Sudden Death..	1	1
Total	10	2

Mortality. In the control group of 22 cases, 1 patient died in a traffic accident and 10 patients (44.4%) died of the effects of coronary artery disease. Following the initial acute attack of myocardial infarction, 3 patients died within 12 months, 3 died in the second year, 3 in the third year and 1 in the fourth year. The causes of death were congestive cardiac failure in 5 patients, acute coronary thrombosis in 4, and acute cardiac failure of undetermined nature in 1 patient.

In the treated group of 29 cases, 2 patients (6.8%) died, both within 1 year of the commencement of therapy. The causes of death were congestive cardiac failure in 1 patient and acute cardiac failure of undetermined nature in the other.

CONTROL OF ANTICOAGULANT THERAPY

Dosage. The weekly dose of dicoumarol necessary to maintain the prothrombin index at about 50% ranged from 250–1,050 mg. with an average of between 300–500 mg. Patients have been divided into 3 groups according to the efficacy of the prothrombin control. Those considered well controlled had prothrombin indices between 40% and 70% for most of the period of treatment, with occasional readings lasting 1 to 2 weeks, above or below these levels. Moderately well-controlled patients were those in whom these variations in the prothrombin index occurred more frequently and tended to be maintained outside the arbitrary therapeutic range for longer than 3 weeks. Poorly controlled patients were those in whom the therapeutic range was difficult to maintain and the prothrombin index varied markedly from reading to reading. There were 17 patients well-controlled, 9 moderately well-controlled and 4 poorly controlled, including 1 well-controlled with dicoumarol but poorly with Tromexan. The series is too small to judge whether the maintenance of an adequate anticoagulant control affected the clinical state of the patient. However, recurrent episodes of acute coronary occlusion and pulmonary embolus did occur in patients who were well-controlled.

Toxic Effects. All patients receiving anticoagulants were warned of the danger of haemorrhage. Of the 8 patients who stopped treatment, 7 did so because of a tendency to bleed. Of those who continued with treatment 11 patients had bleeding of varying severity. In most instances haemorrhage manifested itself as a mild haematuria, epistaxis or easy bruising. In 4 cases haematuria was so profuse as to cause renal colic and in 1 generalized purpura with haematoma formation in the floor of the mouth occurred. In another, following mild exertion, a spontaneous haemarthrosis of the shoulder joint developed. In 3 patients bleeding was post-traumatic and in 2 anticoagulants were discontinued temporarily, in order to obviate haemorrhage prior to dental extraction.

When haemorrhage occurred, dicoumarol was stopped or the dose of the drug reduced. This usually sufficed to stop bleeding but in 6 patients admission to hospital was necessary for treatment.

DISCUSSION

Several reports on the prognosis of coronary artery disease have been published. Owing to the varying periods of observation and to the fact that all patients have not been followed to death, the average longevity, subsequent to an attack of coronary thrombosis, has not been established accurately. In one series,[15] the average survival period of 31 patients who died was 6 months, while in 68 patients who survived, the average survival period was 13.5 months. In another group of cases with coronary artery disease,[16] 1,208 patients had had at least one attack of coronary thrombosis and of these, during a 2-year observation period, 16.9% of the males and 18.5% of the females died. There were 891 patients in whom the first manifestation of coronary artery disease was an acute coronary thrombosis. Of these 423 subsequently died. The average duration of life of those who died was 3.8 years for males and 3.1 years for females, about 46.0% of the males and 58.0% of the females dying within 2 years of the acute attack. In a series of 372 patients[17] who survived the initial episode of myocardial infarction, 101 patients subsequently died. Of these 25.0% died within 1 year and nearly 50.0% within 2 years. Angina pectoris occurred in 63.0% of the cases and congestive cardiac failure developed in over 25.0%. It would appear that, although many patients enjoy long periods of good health following an acute coronary thrombosis, the ultimate prognosis is poor, the average duration of life relatively short and the subsequent development of clinical manifestations of coronary insufficiency common.

While the number of cases in the present series is too small for statistical analysis, certain trends in the course of the disease in the patients treated with anticoagulants continuously as compared with that in the patients not so treated are evident. In the patients who did not continue with dicoumarol, not only was the number of deaths greater but also cardiac failure and recurrent attacks of coronary thrombosis occurred more frequently. Although the incidence of angina pectoris was slightly lower in the control group of patients, a greater number of these died

than did patients on continuous anticoagulant therapy. It is of interest that some of the patients receiving anticoagulants were relieved of angina. In this regard, in a group of patients with angina pectoris alone, not included in this series and receiving dicoumarol continuously, several have experienced either a diminution in the severity and the frequency or a complete abatement of the attacks.

It would appear that the mortality of myocardial infarction and the incidence of its subsequent complications may be lessened when anticoagulants are continued indefinitely. It is possible that by preventing intra-arterial mural thrombus formation, anticoagulants prevent the progression of atheroma and its clinical manifestations of coronary artery disease. However, it is realized that in order to prove the value of this therapy, the observation of a much greater number of patients over a considerable period of time will be necessary.

The control of long-term anticoagulant therapy has been found practicable and relatively safe. In most patients the average maintenance dose of dicoumarol remains constant but in some, for no apparent reason, it may suddenly change. Unless facilities are available for the determination of the prothrombin time and for the immediate treatment of patients who develop toxic effects, the use of this form of therapy should not be undertaken.

SUMMARY

The effects of long-term anticoagulant therapy in a group of 29 patients who recovered from an attack of acute coronary thrombosis are presented. The period of treatment ranged from 2 months to 5 years, the average being 27 months. The number of patients who died or developed subsequent angina pectoris, acute coronary insufficiency, cardiac failure or recurrent episodes of acute coronary thrombosis is noted and compared with a group of 22 patients in whom after recovery from the acute attack anticoagulants were not continued. The mortality and the incidence of cardiac failure and recurrent coronary thrombosis was lower in the treated series.

The toxic effects encountered in the management of these patients are described and the possible significance of the results of therapy discussed.

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PSYCHOSOMATIC ASPECTS OF ULCERATIVE COLITIS

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The treatment of ulcerative colitis has presented a difficult problem in medicine for many years. Few physicians cannot recall the melancholy histories of patients who showed no improvement despite heroic treatment with diet, enemas, sera and infusions until ileostomy became necessary. The mortality rate before the advent of the sulphonamides, antibiotics, cortisone and ACTH has been estimated as 28%,[1] and survival has meant a life of chronic invalidism. Recent work has shown that chemotherapeutic agents are efficacious for the infectious complications of the disease and that cortisone and ACTH may produce a clinical remission in the early stages. However, relapses are common after treatment is discontinued, and it is emphasized that administration of these drugs does not constitute a cure but a valuable adjunct to other forms of therapy.[2] The importance of surgery in long-standing cases in view of the likelihood of neoplastic change has been stressed in a recent paper.[3]

Invasion of the bowel by micro-organisms[4] and enzymatic digestion of the colonic mucosa[5] have been cited as possible causative factors, but no definite aetiology has yet been established. Lium postulated that sustained colonic hyperfunction was responsible for ulceration and produced colonic lesions experimentally with drugs,[6] but how this state of hypermotility comes about in man remained to be explained.

Twenty years ago Murray was the first to describe the relationship between emotional states, personality and the onset and exacerbations of ulcerative colitis.[7] His findings have been corroborated by many subsequent studies.[8-11] Work by Grace, Wolf and Wolff has shown that in a setting of conflicts with anxiety and resentment, the colon is hypermotile, hyperaemic and engorged, fragile and susceptible to injury, so that the otherwise harmless constituents of normal stools, enzymes and bacterial flora, may bring about injury.[12] It is believed that the initial changes in the colon are mediated by impulses from the hypothalamus acting through the sacro-pelvic nerves of the sacral parasympathetic outflow.[13] The authors emphasize the emotional background of the con-

dition. It would thus seem that further study along psychological lines promises to provide the most helpful information for an understanding of the aetiology, course and treatment of the disease.

CASE MATERIAL

Case 1. Mrs. M. K. M., aged 32, married 5 years with one child, had a 10-year history of diarrhoea with blood and mucus diagnosed as ulcerative colitis after sigmoidoscopy and barium enema.

The elder of 2 children, she remarks her resemblance to her father, described as shy, submissive and meticulous. Her mother is a dominant energetic person who has always aided the patient in times of illness. The patient herself is tense, anxious and inhibited, lacking in confidence and initiative and over-dependent on her family.

She was 22 when diarrhoea began 4 weeks before her first marriage. Her fiance was due home from the R.A.F. and she dreaded his return for the marriage as she was no longer in love with him but felt that she had to go through with it for her parents' sake. Symptoms were present on her honeymoon. Diarrhoea increased to 4–5 stools daily on her husband's return to operational flying and became acute when he was reported missing 6 months later.

During the next 3 years her condition gradually improved with medication and she was able to return to work. In 1946 she met her present husband and continued to see him although she felt guilty as he was engaged in divorce proceedings. At this time she began passing 4–5 loose stools daily with blood and mucus. After a month's treatment in hospital she was well enough to marry. She remained well throughout her pregnancy and until the child developed severe whooping cough and measles at 18 months. Her stools were again frequent and offensive and remained so for the next year despite medical treatment. She was living with her in-laws and continued friction made her anxious and depressed. There was a dramatic improvement in January 1951 with the gain of a stone in weight when she moved to her own house. She remained well until February 1952 when her 4-year-old daughter was found to have astigmatism. The patient then developed colicky abdominal pain, frequent stools, and began to lose weight. She also became irritable and depressed, wept easily and was unable to do her housework.

At this stage psychotherapy was begun, consisting of weekly interviews of one hour during which a degree of rapport was established, thus enabling the patient to express her feelings with more confidence. Each session was concerned with a discussion of an important topic, such as the key figures in her life and those periods when symptoms were severe. Several features emerged in therapy, notably her inability to express resentment, her fear of reprisal if she should become aggressive, as she put it: 'I haven't the guts for it.' Her attitude towards her mother was ambivalent. She was extremely dependent on her for advice and support but at the same time resented her officious, authoritarian approach and her lavish presents to her granddaughter—in marked contrast to the patient's own tendency to stinginess. Her main anxiety, however, appeared to be over her child. She felt personally responsible for the child's poor eyesight. While pregnant she had fantasies of contamination of the uterus by rectal contents during intercourse, and the first question she asked after the child's delivery was: 'Has it got colitis?' Her belief was strengthened by being told that an attack of cystitis during pregnancy was due to 'a germ from the bowel getting into the bladder'. At a later session she reported an acute recurrence of colicky abdominal pain and diarrhoea after her daughter had again seen an oculist who said that treatment might be necessary. She immediately thought of an operation and abdominal cramps began an hour later.

After 18 interviews the patient reported that she was feeling less anxious and depressed, was having one semi-formed stool daily without blood or mucus and had gained a stone in weight. She was sleeping well, her appetite was good, she was less fearful about her daughter's health, and now intended to have another child. When seen again 3 months later her improvement had been maintained.

Case 2. Mrs. H. L. R., aged 25, married for 3 years, was admitted to the Maudsley Hospital in July 1949 under the care of Dr. Denis Leigh, with a 3-year history of ulcerative colitis. Her symptoms began 2 months after marriage, lasted for 3 months and cleared up when she returned to live with her mother. The disease followed an intermittent course during the next 3 years, the longest period of freedom from symptoms being 5 months.

The patient had always been an apprehensive person, very dependent on her mother, prone to shirk unpleasant tasks and reacting to periods of stress with marked anxiety. She had had a strict religious upbringing in a family which frowned upon emotional expression in any but the mildest forms.

Psychotherapy brought out the fact that her marriage and consequent separation from her parents precipitated the onset of ulcerative colitis; it became clear that each subsequent separation from her mother occasioned a further attack. She gained insight, but still remained inhibited and unable to express herself. After discharge, therapy was continued on an out-patient basis and she became increasingly able to express feelings of aggression towards her mother without anxiety and with a corresponding improvement in symptoms. She gave birth to a child in June 1951. During her pregnancy there was a recurrence of symptoms lasting a week. Two months after the birth symptoms reappeared. She was very anxious about the baby, but the main conflict was over her mother's possessive attitude to the child and her own desire to accept the responsibility of motherhood, coupled with her inability to do so without her mother's help. This situation was discussed in a further series of 6 interviews, and her stools were reduced to 3 daily. She has remained well up to the present, stools fluctuating between 2 and 3 daily with occasional blood and mucus, but she is able to carry on her work and care for her child.

Case 3. Mr. S. P. (Jewish), aged 28, was seen as an out-patient at the Maudsley Hospital, in August 1950, by Dr. Denis Leigh. He had a 5-year history of diarrhoea beginning when he was in the army in Germany. After investigation a diagnosis of Crohn's disease was made and he had an operation to exclude the terminal ileum. His symptoms became much worse after the operation. He lost 3 stone in weight and was having 12 stools daily. Sigmoidoscopy showed evidence of ulcerative colitis.

The patient was an only child and had been pampered by an over-possessive mother during childhood and adolescence. He had always been a reserved person with limited interests and no strong ambitions. Analysis of the time relationship of symptoms revealed that acute phases were associated with periods of stress, e.g. leaving the army, writing an examination, engagement, marriage and finally the operation. After 5 months of therapy, consisting of weekly interviews during which these stress situations and his hostility to his mother were discussed, there was a slight improvement in his condition.

(This case is of interest because of the association of Crohn's disease with ulcerative colitis.[14])

Case 4. Mrs. M. C. C., aged 39, married 11 years with no children, was admitted to the Maudsley Hospital, under the care of Dr. Denis Leigh, in June 1950, with a 13-year history of intermittent vomitting and diarrhoea with blood and mucus, averaging 8 or 9 stools daily during severe attacks. A diagnosis of ulcerative colitis was made in March 1950.

The seventh in a family of 9, she was handicapped by severe myopia since childhood. Over-dependent first on her mother and then on her husband, she was a sensitive, submissive, inhibited person, meticulous about housework and personal cleanliness, resistant to changes in routine, obstinate and parsimonious. She married at 28 and had an unsuccessful sexual adjustment.

A close relation between stressful situations and fluctuations in the course of illness was established. The first attack of vomiting and diarrhoea occurred in June 1937, when she became engaged in the face of her mother's opposition. The next severe attack took place 2 weeks after marriage in March 1938, when she was particularly upset about her mother's refusal to attend the wedding. In December 1941 she was worried about her husband in the R.A.F. and diarrhoea and vomiting returned for 3 months. She then remained well until June 1947 when constant quarrels with her neighbours began and her symptoms recurred. After several admissions

to hospital for investigation in the next 3 years, a diagnosis of ulcerative colitis was made during a severe attack of diarrhoea which followed a period of worry about her brother who had had pneumonia.

After a full investigation and confirmation of the diagnosis, the patient was given modified insulin therapy while the psychiatrist attempted to establish a relationship with her as the basis for a discussion of her problems. Her symptoms improved over the next 5 weeks, but as psychotherapy continued she became increasingly hostile to the nursing staff and the therapist. Her physical condition deteriorated markedly with a return of diarrhoea and a loss in weight. She eventually took her discharge against medical advice.

Case 5. Mrs. J. C., aged 25, was admitted to the Maudsley Hospital under the care of Dr. Denis Leigh in May 1952 with complaints of depression, insomnia, colicky abdominal pain and frequent loose stools with blood and mucus. A diagnosis of granular procto-colitis had been made after a full investigation in April 1952, but her condition had deteriorated in spite of treatment and on admission there was evidence of anaemia and marked weight loss. A diagnosis of ulcerative colitis was made and medical therapy instituted, followed by psychotherapy a few days later.

She was the second youngest child in a closely-knit middle class family of 6 children. Her quiet, passive father apparently played an insignificant role. Her mother, an active, aggressive and dominant personality, was over-protective towards her children and particularly towards the patient, who was least able to fend for herself. She had always smoothed over the patient's difficulties and made decisions for her.

After an uneventful childhood, she was married at 24. Her family was opposed to the match, regarding her husband as neither suitable nor stable, and there had always been friction between her mother and husband, the latter resenting the former's domineering, possessive attitude. The husband himself was an insecure, nervous individual, unable to satisfy the patient's needs for protection and attention, and she found the break with home and the responsibility of housekeeping and marriage a great strain.

Detailed investigation of symptoms revealed that she had suffered from occasional abdominal cramps since October 1951 when she saw her husband having an 'epileptic fit'. She became extremely anxious after this, fearing that it might happen again. The 'fit', moreover, confirmed her fear that her husband was a 'nervous' person and supported her mother's veiled accusations against him. The mother became even more outspoken in her criticisms of the husband, causing the patient much guilt and anxiety. Since January 1952 she had been passing bright red blood in stools, but attributed it to haemorrhoids. In April 1952, following confinement to bed with influenza, diarrhoea began 4–5 times daily with blood. She described her resentment at being left alone by her husband, and also that her mother did not visit her but went to her brother instead. She felt a strong desire to go home to her mother's care 'where I could get proper food and attention'. This was done, but there was no improvement in her symptoms.

Daily psychotherapeutic interviews commenced with a discussion of her family situation. At first there was an improvement in her symptoms, but as deeper feelings towards her parents and husband emerged the patient became hostile and resentful. Her symptoms became very severe and psychotherapy was discontinued in favour of a medical regime. Eventually her condition deteriorated so badly that an ileostomy had to be performed.

DISCUSSION

1. *Personality Structure*: Previous writers on this subject have all commented on certain personality traits commonly present in patients suffering from ulcerative colitis. These characteristics include, among many others: emotional immaturity with over-dependence on parental figures and other relatives,[7-9, 11] over-conscientiousness and a frequent sense of neatness,[9, 11, 12] emotional tension,[9, 12] suppressed hostility and inability to express aggressive feelings with conflict and guilt.[11, 12]

A study by Mahoney, Bockus *et al.* of 20 patients using the combined method of psychiatric interview and Rorschach testing concluded, as does Wittkower,[13] that the patients presented an abundance of traits commonly observed in the neuroses and other psychosomatic disturbances, but that none of the personality traits found in ulcerative colitis when taken alone is specific for the disease,[16] a point further emphasized by Groen and Bastiaans.[17]

2. *Relation to Changes in Life Situation*: Prugh submits the following positive diagnostic criteria which must be satisfied before a psychogenic cause for ulcerative colitis can be accepted: the presence of an unresolved emotional conflict, the operation of a precipitating event or set of circumstances, and the appearance of a partial solution of the conflict as a result of the persistence of gastro-intestinal symptoms.[18]

Certain significant events, in some cases occurring within 48 hours of an attack, have been found to be closely related to the onset and acute exacerbations of the disease.[15] For example, the loss of a key person in the environment, commonly a parent, sibling, or fiance, either by death, separation, or even an action interpreted by the patient as a loss of love or affection. The mental state in this case is often that of a morbid grief reaction.[19] Other incidents include: a surgical procedure[10]; events giving rise to severe stress, e.g. pregnancy or childbirth[9]; conflicts over dependence and independence, e.g. marriage, and sexual conflicts. The close time relationships indicate the important effect of such events on the condition, but it is uncertain whether emotional conflicts are responsible for each attack or whether there are additional precipitating factors.

3. *Effect of Psychotherapy on the Disorder*: Current opinion seems to favour the 'superficial' or supportive, as opposed to the more 'extensive' or psychoanalytic type of therapy. Lindemann warns against efforts at intensive exploration because of 'the possibility of psychotic-like episodes of primitive behaviour and by intensification of somatic delusions or paranoid manifestations'.[19] The stirring up of emotional conflicts in this way may be followed by a severe or even fatal aggravation of symptoms.

In the preferred method the physician by means of frequent interviews, establishes a relationship with the patient who provides a detailed life history from his childhood to the present. Close attention is paid to relationships with parents and siblings. The physician's attitude is one of interest, encouragement and support. As the patient becomes more sure of the physician's uncritical attitude, deeper feelings of hostility, anxiety and inferiority emerge which the patient is encouraged to express. The ability to voice feelings of resentment is particularly important as personality studies of patients have shown that the inability to do this is a constant feature resulting in suppressed resentment and a tendency to 'stew over' things. Some authors maintain that the symptoms are an expression of suppressed hatred.[7] The opportunity to give vent to these emotions before an uncritical listener results in a diminution of anxiety, guilt and tension, with an increase in confidence and an amelioration of symptoms.

Groen and Bastiaans, using a supportive type of therapy in 29 cases of moderate to severe ulcerative colitis followed over a period of 1–10 years, record remission lasting at least a year in 14 cases, one or more recurrences after initial cure in 6 cases, all of which yielded to further treatment, and an improvement in 7 cases.[17] Grace, Wolf and Wolff, employing interview therapy and a constructive physician-patient relationship in 19 unselected cases of ulcerative colitis over a period of 6 months to 2 years, report considerable improvement in 11 cases, slight improvement in 2 cases, and no improvement in 6 cases.[12]

SUMMARY

A brief review has been given of the literature concerning the psychosomatic aspects of ulcerative colitis, together with case reports of 5 patients treated by psychotherapy. The findings insofar as personality traits, precipitating factors and the relationship of the course of the disease to life situations, bear a marked similarity to the conclusions of previous studies. The success of psychotherapy in certain cases would seem to confirm the need for further work in this field, particularly in the early stage of the disease.

I am indebted to Dr. Denis Leigh, Physician to Bethlem Royal and Maudsley Hospitals, under whose supervision these patients were treated, for permission to publish the case records.

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ABSTRACTS

C. S. Webster. *Non-Surgical Repair of Cystocele and Rectocele: An Original Technique.* (*Arizona Medicine*, 1952, **9,** 27.)

Cystocele and rectocele are among the most common pathological conditions resulting from childbirth. For many reasons scores of women cannot or will not submit to surgical treatment. The author, having observed the satisfactory results of the treatment of internal haemorrhoids with a sclerosing agent, decided to employ the same principle in the correction of cystocele and rectocele.

Two ml. of a 5% solution of quinine and urea hydrochlori with 2% procaine are injected into the submucous layer of the vaginal wall. The usual method is to inject one ml. into the right side and one ml. into the left side of the anterior wall; the second treatment can be into each lateral wall; the third into the posterior wall; then proceed at different levels. If the patient does not experience more than a momentary dizziness the dose can be, and usually is, increased to 2 ml. for each injection at the next visit: a total of 4 ml. per treatment. These are given twice weekly. An average case requires approximately 50 ml.

Topical anesthesia, such as 2% pantocaine, is swabbed over the site of injection. A three-inch No. 22 guage Goldbacher needle is very satisfactory. If the tissue turns white at the point of insertion the needle must be immediately withdrawn to avoid sloughing. This is particularly likely to happen on the anterior wall, which frequently is leather-like and cracked. Poorly nourished as this tissue is, it is amazing how quickly a slough will heal after the introduction of a tampon covered with a soothing ointment. The patient is often unaware of it, and, fortunately, under any circumstances, it is merely a minor discomfort. Sloughs will occur in spite of the utmost care. The needle should remain *in situ* for about a minute.

A tampon lubricated with a soothing, astringent ointment is packed high in the vaginal vault and one or two smaller ones into the lateral fornices. The tampons are retained for two or three days if possible, and after removal are followed by a soothing douche.

Time, of course, must be allowed for the full effect of the shrinkage, although distinct improvement not infrequently appears after three or four treatments. There are many women in the fourth and fifth decades of life who are decidedly uncomfortable because of relaxed vaginal walls only, and who can be greatly relieved by a few injections into the billowing tissues. Occasionally a patient returns after months or years for a few additional treatments.

Dr. Webster's paper concludes with five illustrative case reports. Her conclusion is: 'A sclerosing agent injected into the submucous layer of the vagina will produce contraction of the relaxed walls and thereby support the prolapsed organs involved in cystocele and rectocele.'

J. G. Herschell. *The effect of B.S. 572 on Intermittent Claudication.* (*Geneeskundige Gids*, 25 January 1951, p. 26.)

Following a favourable effect of cyclospasmol having been noticed upon a patient who suffered from the disease of Bürger, the relative remedy was administered to those suffering from spastic contractions of the peripheral vessels, *inter alia* of an arteriosclerotic nature. The oxygen supply of the muscles would be inadequate should these patients do any additional work; however, both this insufficiency and the accompanying pains disappear directly the muscle is in rest again. In this way it can be tried to bring about progress through the relative substance in cases of intermittent claudication; a description is given of the efforts made in eight cases. The author mentions the tensiographic results, stating that he is convinced that a favourable effect should be ascribed to cyclospasmol upon the aforementioned disorders which is also evident from the significant improvement of the tensiogramme of the arteries. The author points out that he has never seen this improvement of the tensiogramme following the administration of remedies previously used. Within a short time the subjective improvement becomes evident, but the objective improvement which is shown by the tensiogramme can only be noticed much later, so that it is surely necessary to continue the treatment during some months. The normal dose is one tablet of 20 mg. to be taken three times a day. Undesirable effects have never been noticed.

South African Medical Journal
Suid-Afrikaanse Tydskrif vir Geneeskunde

EDITORIAL

A FRIENDLY GESTURE FROM ITALY

All South Africans will be interested in a recent issue (24 February 1953) of *Minerva Medica*, the Turin Journal of Practical Medicine. This issue, comprising 52 pages of text, is exclusively devoted to South African authors. In fact, it might well be an issue, translated into Italian, of a South African medical periodical. It is published under the general title 'Medicine in South Africa' (*La Medicina in Sud Africa*) and is compiled under the direction of Professor Guy A. Elliott and Professor Joseph Gillman of the Witwatersrand University, Dr. Eric Samuel of Johannesburg and Dr. Adolphe Shedrow, President of the South African National French Association.

The issue opens with cordial messages of international goodwill from our Minister of Health (Dr. Karl Bremer, M.P.), the Italian Minister Plenipotentiary in South Africa (Marchese Gustoforo Fracassi) and the Honorary President of the South African French Association (Sir Ernest Oppenheimer). It contains two articles by Professor Elliott on Races and Disease in the Union of South Africa, and Coronary Disease in the Bantu. Dr. L. Fatti of Johannesburg General Hospital writes on the Surgery of Mitral Stenosis, and Drs. J. Gear and A. Zoutendyk of the S.A. Institute for Medical Research, on Auto-Antibodies and Auto-Allergic Diseases. There is a report by J. Gillman, C. Gilbert, T. Gillman and I. Spence of a research on the Relation of Maize to Diseases of the Liver, conducted under the auspices of the Council for Scientific and Industrial Research and the Witwatersrand University. Drs. J. C. Gilroy, P. Marchand and V. H. Wilson of Baragwanath Hospital contribute an article on the Blood Circulation of the Lungs in Pathological and Normal Conditions; Dr. A. Rowland Krynauw and H. D. Ritchken of Johannesburg General Hospital, on Hemispherectomy; and Dr. Shedrow, on a Radiological and Clinical Study of Chronic Amoebic Colitis.

Thus South African Medicine on this occasion is represented exclusively by Johannesburg, apart from the abstracts, which this issue of *Minerva Medica* contains, of 14 articles published in 1951 and 1952 in the *South African Journal of Clinical Science* and the *South African Medical Journal*, and one in the *South African Journal of Medical Sciences*. The representation is well worthy of the occasion and the medical profession throughout South Africa will be grateful to those colleagues who have contributed of their work.

It is our duty and pleasure to offer our hearty thanks to the *Minerva Medica* and to recognize with gratitude this

VAN DIE REDAKSIE

'N VRIENDSKAPSGEBAAR VAN ITALIË

Alle Suid-Afrikaners sal belangstel in 'n onlangse uitgawe (24 Februarie 1953) van *Minerva Medica*, die Turynse Tydskrif vir Praktiese Geneeskunde. Hierdie uitgawe, wat 52 bladsye van teks beslaan, is uitsluitlik aan Suid-Afrikaanse skrywers gewy. Dit kon feitlik 'n uitgawe van 'n Suid-Afrikaanse mediese tydskrif, wat in Italiaans vertaal is, gewees het. Dit is onder die algemene opskrif 'Geneeskunde in Suid-Afrika' (*La Medicina in Sud Africa*) gepubliseer en onder die toesig van professor Guy A. Elliott en professor J. Gillman van die Witwatersrandse Universiteit, dr. Eric Samuel van Johannesburg, en dr. Adolphe Shedrow, President van die Suid-Afrikaanse Nasionale Franse Vereniging, saamgestel.

Die uitgawe begin met hartlike boodskappe van internasionale welwillendheid van ons Minister van Gesondheid (Dr. Karl Bremer, L.V.), die Italiaanse Gevolmagtigde Minister in Suid-Afrika (Markies Gustoforo Fracassi) en die Ere-President van die Suid-Afrikaanse Nasionale Franse Vereniging (Sir Ernest Oppenheimer). Dit bevat twee artikels deur professor Elliott, een oor rasse en siekte in die Unie van Suid-Afrika en die ander oor kroonaaraandoenings by die Bantoe. Dr. L. Fatti van Johannesburg se Algemene Hospitaal skrywe oor die chirurgie van myterstenose, en drs. J. Gear en A. Zoutendyk van die S.A. Instituut vir Mediese Navorsing oor outoteenstowwe en outo-allergiese siektes. Daar is 'n verslag deur J. Gillman, C. Gilbert, T. Gillman en I. Spence van 'n navorsing oor die verhouding van mielies tot lewerkwale, wat onder beskerming van die Wetenskaplike en Nywerheidsnavorsingsraad en die Universiteit van Johannesburg uitgevoer is. Drs. J. C. Gilroy, P. Marchand en V. H. Wilson van die Baragwanath hospitaal dra 'n artikel oor die bloedsomloop van die longe onder patologiese en normale omstandighede by; drs. A. Rowland Krynauw en H. D. Ritchken van die Johannesburgse Algemene Hospitaal oor hemisferektomie; en dr. Shedrow oor 'n radiologiese en kliniese studie van kroniese amebiese dikdermontsteking.

Met hierdie geleentheid word Suid-Afrikaanse Geneeskunde dus uitsluitlik deur Johannesburg verteenwoordig, met uitsondering van uittreksels van 14 artikels wat in 1951 en 1952 in die *Suid-Afrikaanse Tydskrif van Kliniese Wetenskap* en die *Suid-Afrikaanse Tydskrif vir Geneeskunde* gepubliseer is, en een artikel wat in die *Suid-Afrikaanse Tydskrif van Mediese Wetenskappe* verskyn het. Die verteenwoordiging is die geleentheid wel waardig, en die mediese professie dwarsdeur Suid-Afrika sal dankbaar teenoor daardie kollegas wees wat van hulle werk bygedra het.

Dit is ons plig en plesier om ons hartlike dank teenoor die *Minerva Medica* uit te spreek, en met dankbaarheid hierdie merkwaardige betoning van welwillendheid van

notable expression of the goodwill of Italy and its medical profession towards our own country and profession. It is a manifestation of the international solidarity of Medicine which indeed 'knows no frontiers'. We particularly value it as coming from that country and people who were in the van of the Renaissance and have ever been in the forefront of the Arts and Sciences and of Medicine. We greet our Italian colleagues, and we offer our salutations to their great nation.

Italië en sy mediese professie teenoor ons eie land en professie te erken. Dit is 'n openbaring van die internasionale eenheid van geneeskunde wat inderdaad ‚geen grense ken nie'. Ons waardeer dit, veral as komende van daardie land en volk wat die voorhoede van die Renaissance gevorm het en steeds op die voorpunt van die Kuns, die Wetenskappe en die Geneeskunde bly. Aan ons Italiaanse kollegas groete en aan hul groot volk ons saluut!

BORNHOLM DISEASE, PLEURODYNIA OR EPIDEMIC MYALGIA

AN OUTBREAK IN THE TRANSVAAL ASSOCIATED WITH COXSACKIE VIRUS INFECTION

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Middelburg, Transvaal
VERONICA MEASROCH, Ph.D.
JAMES GEAR, M.B., Ch.B.
South African Institute for Medical Research, Johannesburg

This paper reports an epidemic of an illness, characterized by the signs and symptoms of Bornholm disease, which occurred in Middelburg, Transvaal, during the latter half of January and the first week of February 1952. From a significant number of cases a virus identified as Coxsackie Group B type 4 virus was isolated.

The epidemic affected the children living in a school hostel. Of 180 children in the hostel, 40 (22%) developed the illness. The first case probably occurred on 19 January 1952, but the identity of the disease was only defined on 27 January 1952, after 5 cases had presented themselves. New cases were reported till 6 February 1952, with the peak on 27 January (5 new cases) and 28 January (6 new cases). By 8 February all the children were back at school and no further cases occurred. The outbreak was confined to hostel children who all attended the same school as 150 day scholars amongst whom no cases occurred. Included in the 40 cases is one boy who attended the same school but lived in another hostel. This boy was, however, in close contact with the hostel boys and spent his afternoons at the affected hostel, so that he too was virtually an inmate of the affected hostel. Although he was in close contact with 70 other boys at his hostel, no further case was seen from that hostel.

CLINICAL FINDINGS

The illness was sudden in onset. Rigors occurred in 4 cases (10%), but in 36 (90%) patients the illness presented with sudden acute epigastric pain. The pain radiated across the upper abdomen, but was usually more severe in the right hypochondrium. In 4 of these cases the pain radiated into the chest but was not severe. The pain was either stabbing or cramping in nature and was aggravated by movement, breathing, coughing, laughing, crying or sneezing. In 13 cases (32.5%) the pain was accompanied by nausea and vomiting. In 2 (5%) a mild diarrhoea occurred. In 4 (10%) the pain radiated to the lower abdomen, and in 2 of these localized in the right iliac fossa simulating acute appendicitis. Four cases (10%) had chest pain only and this was pleuritic in character. In 2 the pain occurred across the whole of lower chest while in the other 2 the pain localized in left chest. One case developed a transient pleural friction rub. In a further four cases the chest pain and abdominal pain were of equal intensity. In this outbreak therefore, 36 cases (90%) presented with some form of abdominal pain, which was the prominent symptom. The only prominent physical sign was epigastric tenderness and tenderness in hypochondria, especially along the subcostal margins. Voluntary guarding occurred in several cases, but rigidity and rebound tenderness were not encountered. The breathing was shallow but not rapid and in the more severe cases the patient lay absolutely still with his hand clasped to the affected side to prevent unnecessary movement.

Pyrexia occurred in 32 cases (80%) and varied from 99°F to 105°F but was usually between 100–102°F. The pulse was usually raised proportionately but in several cases the high temperatures were accompanied by a slow pulse rate.

Headache was a prominent symptom, occurring in 22 cases (55%). The illness often presented with headache and was soon followed by abdominal or chest pain. The headache was frontal in situation and in several cases was very severe, overshadowing other symptoms.

Backache only occurred in one case (2.5%). Shoulder tip pain occurred in 4 cases (10%). In one case the illness commenced with hiccoughs. Dizziness was complained of by 4 patients (10%) but meningismus or neck pain did not occur in any of the cases.

Sore throat occurred in 3 cases (8.5%) and in these cases there was slight faucial congestion. Cough did not occur in any of the cases, and except for a pleural friction rub in one case, no lung signs were found.

In one third of the cases, a relapse occurred after an interval of 2–3 days. In 4 cases 2 relapses occurred. The relapses duplicated the original symptoms almost identically, and in most cases were of equal severity.

Improvement in the pain was parallel to defervescence but abdominal tenderness persisted for about 48 hours after all symptoms had disappeared. No case was regarded as cured till abdominal tenderness had disappeared.

The duration of illness, i.e. till all signs of abdominal tenderness had disappeared, varied from less than 24 hours to 11 days with an average of 4.5 days.

Although only 5 adult cases were seen during this period, it is interesting to note that in 4 chest pain of very severe type occurred, and in 2 of the cases pleural friction rubs were heard; in one case the friction rub was heard over a large area on both sides. Only one case presented with severe epigastric pain simulating a perforate peptic ulcer but no rigidity or guarding was present. It therefore appears that in children the disease presents mainly with abdominal symptoms—abdominal pain, nausea, vomiting and occasionally diarrhoea, but gastro-intestinal symptoms are not so prominent in adult cases, in which chest signs and symptoms are more obvious. It seems then that, in children, the name often given to Bornholm disease, viz. epidemic pleurodynia may be misleading.

In the present outbreak the diagnosis was seldom in doubt, but the 2 cases presenting as acute appendicitis could have been regarded as surgical conditions had they occurred sporadically. The white cell count is not of much help in early cases but in our cases the tenderness was superficial, rigidity and rebound tenderness were absent. However, cases of Bornholm disease are often operated on for abdominal surgical conditions, usually acute appendicitis. This is likely to occur in children rather than in adults.

The characteristic clinical picture is illustrated by the following cases:

Case 1: L. B. Age 10. Female.

21 January 1952. Took ill suddenly at school at about 10 a.m. with severe pain in left chest anteriorly, aggravated by movement, breathing or crying. No cough. Temperature 102°F. Pain slowly subsided over period of 6 hours. Had a mild headache.

24 January 1952. Quite well for 2 days then suddenly developed identical pain but this time it radiated to left shoulder tip as well. No cough. Slightly dyspnoeic. Temperature 102°F.

25 January 1952. Apyrexial. Pleural rub left chest anteriorly.

26 January 1952. Patient quite well. No further relapse.

Case 4: G. B. Age 10. Male.

26 January 1952. Soon after breakfast patient developed hiccoughs, which lasted about ½ hour. A few minutes after onset he developed acute upper abdominal pain, stabbing in nature and was forced to lie down. Walking was impossible as this aggravated the pain. In the afternoon he had a short rigor and felt very hot. By evening there was no pain and no cough.

Temperature 101.6°F. Pulse 116 per minute. Respiration 20 per minute. Tender in upper abdomen, especially subcostally in both sides. No guarding. No rigidity.

27 January 1952. No pain. Apyrexial. Still tender.

28 January 1952. Quite well.

29 January 1952. Patient was allowed out of bed. Felt quite well. About 2 p.m. he suddenly developed aching pain in right hypochondrium. Aggravated by deep breathing. Temperature 101°F. Pulse 80 per minute. Respiration 22 per minute. Tender in epigastrium and right costal margin.

30 January 1952. All pain gone but slight pyrexia. Abdominal tenderness still present. Temperature 99°F.

1 February 1952. N.A.D.

2 February, 1952. Temperature 101°F. Otherwise feels well.

3 February 1952. Sudden recurrence of pain in epigastrium and right hypochondrium. Tender in right hypochondrium and right iliac fossa.

4 February 1952. Apyrexial. Tender along costal margin.

5 February 1952. Apyrexial. N.A.D.

The test for Coxsackie virus in suckling mice gave a positive result.

Note: 2 relapses (Fig. 1).

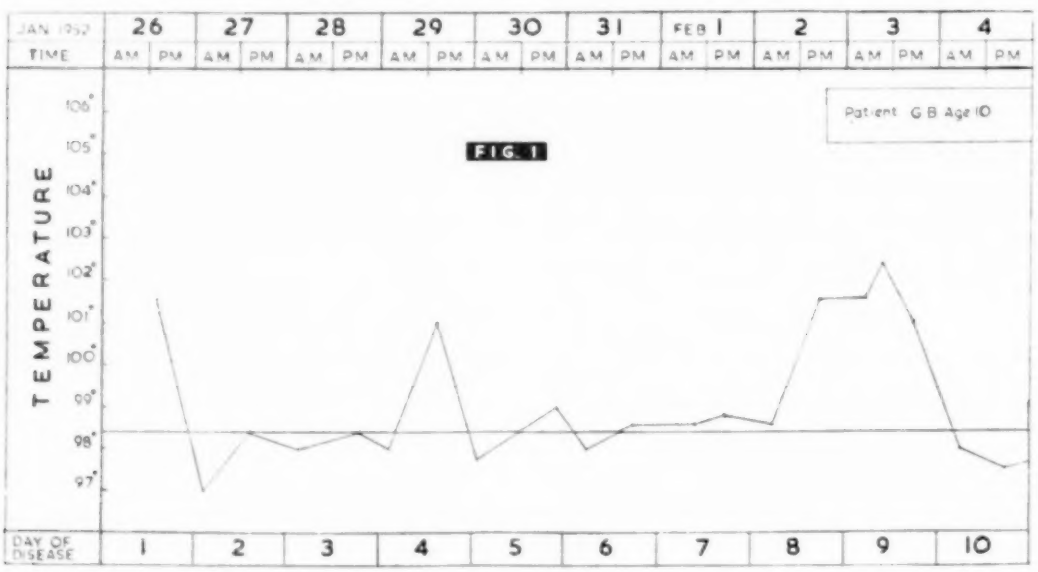

Fig. 1 (Case 4). Note 2 recrudescences after the initial attack of fever.

Case 5: G. S. Age 10. Male.

22 January 1952. Soon after lunch patient developed upper abdominal pain, which later localized to epigastrium and was aching in character. At same time he developed 'shivers' and vomited once. No diarrhoea. No cough.

23 January 1952. Pain in epigastrium still present but felt better. Tender along costal margin and epigastrium.

26 January 1952. Quite well.

28 January 1952. Return of pain. Aggravated by deep breathing. Temperature 99.6°F. Pulse 88 per minute. Respiration 18 per minute.

29 January 1952. Quite well.

Test for Coxsackie virus positive.

Case 9: N. B. Age 6. Male.

27 January 1952. Sudden onset of upper abdominal pain. Aggravated by laughing, breathing or movement. No vomiting. No diarrhoea. Later in day pain moved to right iliac fossa. Seen at 6 p.m. Tender in upper abdomen, especially under ribs. Tender in R.I.F. No guarding. No rigidity. Temperature 101.4°F. No headache.

28 January 1952. Patient much better. No pain. Temperature 99.4°F. Still tender in upper abdomen.

29 January 1952. Sudden recurrence of identical pain as on 27 January 1952. Temperature 102°F. Pulse 118 per minute. Respiration 20 per minute. Tender in upper abdomen and right iliac fossa.

30 January 1952. Patient much better. Apyrexial. Still tender in upper abdomen.

31 January 1952. Recurrence of pain again. Temperature 101°F.

1 February 1952. Pain better. Temperature 99.4°F. Pulse 74 per minute. Respiration 20 per minute.

2–3 February 1952. No pain, but tenderness present.

4 February 1952. N.A.D.

Note: 2 relapses (Fig. 2). Test for Coxsackie virus positive.

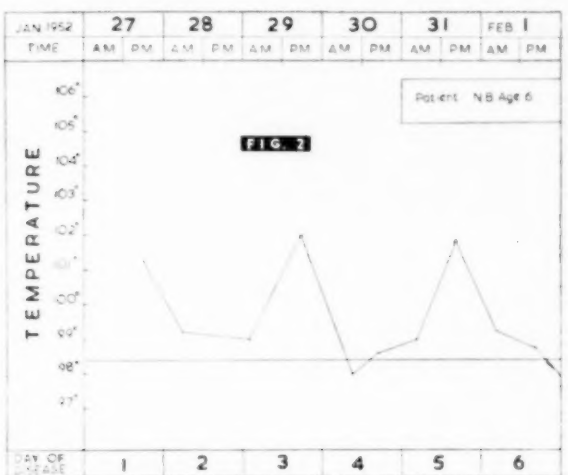

Fig. 2 (Case 9). Note 2 recrudescences of fever.

Case 10: R. G. Age 7. Male.

27 January 1952. Sudden onset of pain in right lower chest and right upper abdomen. Aggravated by breathing and crying. Felt feverish and had rigor. No headache. No cough. No vomiting or diarrhoea. *6 p.m.* Tender in right hypochondrium. Painful breathing.

28 January 1952. No pain. No tenderness. Apyrexial.

29 January 1952. Recurrence of abdominal pain but less severe than previous attack. Temperature 98.8°F. Pulse 70 per minute. Respiration 24 per minute. Abdomen tender in right hypochondrium.

30 January 1952. Patient feels well. Apyrexial. Abdomen still tender.

31 January 1952. N.A.D.

Test for Coxsackie virus positive.

ANALYSIS OF SYMPTOMS IN 40 CASES OF BORNHOLM DISEASE

Symptoms	*No. of Cases*
Abdominal pain	32 (80%)
Abdominal pain and chest pain of equal intensity	4 (10%)
Chest pain only	4 (10%)
Pyrexia	32 (80%)
Headache	22 (55%)
Vomiting	13 (32·5%)
Relapse (one)	9 (22·5%)
Relapse (two)	4 (10%)
Rigors	4 (10%)
Dizziness	4 (10%)
Sore throat	3 (8·5%)
Diarrhoea	2 (5%)
Shoulder tip pain	2 (5%)
Pleural rub	1 (2·5%)
Backache	1 (2·5%)

LABORATORY STUDY

MATERIAL

Specimens of blood and faeces were collected from a number of the patients. Some were obtained during the acute phase and again in the convalescent phase from the patients in which this was possible. In the patients who had already recovered only convalescent specimens were available. These specimens were sent by passenger train to Johannesburg. On arriving at this Institute the following morning, about 24 hours after collection, the serum was separated from the blood and then the sera and the faeces were stored in a deep freeze refrigerator at −10°C to −20°C pending the laboratory tests for the presence of virus and the detection of serum antibodies.

ISOLATION VIRUS

A 10% suspension of each specimen of faeces was made in broth and 1/4 volume of anaesthetic ether added and allowed to act for 48 hours in a +4°C. refrigerator. The suspension was then spun in an angle centrifuge at 3,000 revolutions per minute for one hour. The clear aqueous phase underlying the ether layer was withdrawn and 0.03 c.c. of this suspension was inoculated subcutaneously into each of a litter of 6–8 baby mice 0–2 days old.

These baby mice were observed for paralysis, tremors, and other signs of illness. When one became weak or paralysed it was killed with ether. The chest and abdominal viscera were removed and a broth extract was made of the carcass and brain for further passage in baby mice.

At the same time specimens of the various organs and tissues were placed in Bouins fixative solution prior to preparation for embedding in paraffin and the cutting of histological sections. The histological sections were stained with haematoxylin and eosin. The results are summarized in Table I.

A virus pathogenic to baby mice was isolated from 9 of 18 specimens of faeces from these patients, collected on 28 January 1952, and from 3 of 9 specimens collected from the positive patients 6 weeks later on 12 March 1952.

The clinical picture in the baby mice was similar in all cases. On primary isolation paralysis or weakness of the limbs often associated with tremors developed from 2–8 days after inoculation. On passage the incubation period became fixed at 2–3 days. Often the paralysis affected only one limb or part of one limb causing wrist drop or foot drop. After developing paralysis the baby mice usually died within 24 hours. Sometimes mice were found dead without having

TABLE I: ISOLATION AND IDENTIFICATION OF VIRUS FROM STOOLS OF PLEURODYNIA PATIENTS

No.	*Patient's Initials*	*Sex*	*Age*	*Virus Isolation*			
				Specimen (28 January 1952)	*Type*	*Specimen (13 March 1952)*	*Type*
1	R.O.	M	6	+	B4	−	
2	G.B.	M	10	+	B4	−	
3	G.S.	M	10	+	B4	−	
4	B.W.	M		+	B4	+	A3
5	K.E.	M	7	−			
6	C.H.	M		+	B4	+	B4
7	N.B.	M	6	+	B4	−	
8	R.G.	M	7	+	B4	−	
9	J.M.	F	12	+	B4	−	
10	A.S.	F	11	+	B4	+	B4
11	K.T.	F	5	−			
12	C.S.	F	11	−			
13	L.W.	F		−			
14	E.T.	F	9	−			
15	C.T.	F	10	−			
16	J.C.	F	9	−			
17	R.L.	F		−			
18	E.W.	F		−			

exhibited weakness the day before. Often the mother ate the dead or dying babies and no trace of them was found at the next checking. Many of the baby mice recovered from their illness and then continued to thrive.

Pathology in Experimental Animals

Histological sections were prepared from the various organs and tissues of infant mice sacrificed at intervals after inoculation. They included sections from mice infected with the strains isolated from several different patients and also serial sections of selected infant mice. A study of these histological sections revealed characteristic lesions in the interscapular fat body. There was often extensive destruction of cells associated with neutrophil and monocyte infiltration. The pancreas in some cases showed extensive disintegration of the structure and dissolution of the parenchymal cells associated with an infiltration of monocytes, neutrophil and occasional eosinophil leucocytes. The islets of Langerhans were not involved in this process and appeared normal.

In some baby mice areas of degenerate cells were seen in the liver, which, as is normal at this age, also showed islands of haemapoietic cells. The muscles of the limbs and trunk showed a few small focal inflammatory lesions. In these areas the muscles showed oedema, fragmentation and cell infiltration. Most skeletal muscles however retained their normal structure.

In most of the baby mice inoculated subcutaneously no lesions were noted in the central nervous system, but in a few, small foci of inflammatory cells were observed in the brain sections.

This virus was also pathogenic to adult mice. In one experiment 6 adolescent mice of ±16 gm. were inoculated with 0.25 c.c. of a 1:10 suspension of infected baby mouse carcass. They fai'ed to gain weight normally. Indeed they actually lost weight; on the sixth day after inoculation their average weight showed a loss of 1 gm.

Histological sections from one of these mice killed on the sixth day after inoculation revealed that the central nervous system was normal except for one small focus of inflammation in the brain. There were a few foci of inflammation in the liver in which the parenchymal cells were degenerate with pyknotic nuclei. The muscles showed a few foci of inflammation with infiltration of mononuclear cells and basophilia of the cytoplasm of the muscle cells.

The spleen showed hypertrophy of the germinal centres of the malpighian corpuscles. Sections of the pancreas revealed that large blocks of pancreatic tissue had disintegrated and been replaced by inflammatory cells, mostly monocytes. There were scattered areas of acinar cells. Some were degenerate with eosinophilic cytoplasm and pyknotic nuclei. The islands of Langerhans were spared in this inflammatory process.

Two monkeys were inoculated intracerebrally with 1:10 suspension of infected baby mouse carcass in broth. These monkeys remained clinically well. One was killed by ether-chloroform anaesthesia on the thirteenth day after inoculation. Sections revealed that all its organs were normal in appearence except the brain and diaphragm. A section through the cerebral hemisphere in the region of the motor cortex revealed round cell infiltration of the meninges and perivascular infiltration and a line of inflammatory cell infiltration in the brain substance. This inflammatory reaction apparently took place in the site of the intracerebral injection. It is not possible to say whether this was a reaction to the foreign matter introduced or whether it was a specific reaction to the virus infection. Sections of the other parts of the brain revealed no pathological changes.

The diaphragm showed a linear area of what appeared to be an infiltration of inflammatory cells, but most of its structure was normal. The pancreas and liver were also normal in appearence as were the other organs and muscles.

Comment

These findings reveal that the virus isolated from the cases in this outbreak has the characteristics of the B group of the Coxsackie or C viruses.

Immunological Studies

Preparation of Antisera

To prepare immune sera for the study of the immunological relationships of the strains responsible for this epidemic, monkeys were immunized against the strain isolated from one of the patients in the Middelburg outbreak, and also against each of Dalldorf's types of Coxsackie virus. The latter type strains were kindly sent to us by Dr. Dalldorf.

Each monkey was inoculated with a 1:10 suspension of mouse carcass in broth; 0.5 c.c. was given intracerebrally, 3 c.c. intraperitoneally and 2 c.c. intramuscularly. A week later each monkey was given 3 c.c. intraperitoneally and 2 c.c. intramuscularly. This was repeated after one week. Two weeks after the last injection the monkeys were bled by intracardiac puncture. The serum was separated from the blood clot and tested by the baby mouse protection tests for its protective properties. Provided its neutralizing power was satisfactory, it was kept for the immunological study.

Baby Mouse Protection Test

In the baby mouse protection test 0.5 c.c. of serum was mixed with 0.5 c.c. of a 1:10 dilution of a suspension of infected mouse carcass containing about 1:1,000,000 infective doses. The mixtures were thoroughly shaken and then allowed to stand for one hour at room temperature. Five baby mice of a litter of 7–8 were then inoculated subcutaneously with 0.03 c.c. of each serum-virus mixture. The remaining 2 or 3 baby mice were inoculated with the virus suspension without the addition of serum to serve as controls of its potency. The virus suspension was also titrated in tenfold dilutions from 1:10 to 1:1,000,000 and each dilution inoculated into a litter of baby mice.

The mice were observed for 14 days for signs of tremors, weakness, paralysis or death.

In the first experiment, the viruses isolated from several different Middelburg patients were tested against the R.O. antiserum prepared against the strain isolated from Richard, one of the patien's in the Middelburg epidemic.

The results follow on the next page.

The negative control baby mice all died within 2–3 days of inoculation.

The strains from the various patients were therefore all neutralized by an antiserum prepared against one of the strains, the R.O. strain chosen as being representative. It was thus presumed that they were all immunologically similar and of the same type.

Strain from Patient	*R.O. Antiserum Survivors Total*	*Interpretation*
1. R.O.	5/5	Positive protection
2. C.H.	4/5	Positive protection
3. G.B.	5/5	Positive protection
4. A.S.	5/5	Positive protection
5. N.B.	5/5	Positive protection
6. B.W.	5/5	Positive protection
7. R.G.	5/5	Positive protection
8. Negative Control	0/5	Negative protection

The R.O. antiserum was then tested against the type strains of group B Coxsackie virus sent to us by Dr. Dalldorf, and Group B type 4 antiserum was tested against the R.O. strain and against its homologous virus.

The results of these tests were:

Type of virus	*Antisera*			
	R.O. Middelburg		*B 4 Dalldorf*	
	Test 1	*Test 2*		
	Survivors Total			
R.O. strain	5/5	5/5	5/5	5/5
B 1 Dalldorf	0/5	0/5		
B 2 Dalldorf	0/5	0/5		
B 3 Dalldorf	0/5	0/5		
B 4 Dalldorf	4/5	5/5		5/5

The baby mice inoculated with the virus in each of these tests for the control of its virulence all became paralysed within 2–3 days.

COMMENT

It will be noted that R.O. Middelburg antiserum protected against the R.O. (homologous) strain of virus and also against Dalldorf's type B 4 virus, but not against Dalldorf's B 1, B 2 and B 3 types. Further, Dalldorf's B 4 type antiserum protected against the R.O. strain of virus and the B 4 type. From these results it was concluded that the strain isolated from the patients in the Middelburg epidemic was of the B group of Coxsackie viruses and was immunologically similar to the type B 4 of Dalldorf. [It is of interest to note that the original virus of this type was isolated from a case of pleurodynia].

DEVELOPMENT OF NEUTRALIZING ANTIBODIES IN THE SERUM OF THE PATIENTS

It remained to be proved that this strain of virus was aetiologically related to the illness experienced by the patients involved in the outbreak. If it was, then antibodies against this virus would be expected to develop in the patients' blood during the course of the illness and from being absent in the acute phase would be demonstrable in convalescence.

Sera from several patients were collected on 28 January 1952, in the acute phase or early convalescence and again on 7 March 1952, after recovery. These sera were tested by the baby mouse protection test against varying tenfold, from 1:10 to 1:10,000, dilutions of R.O. virus suspension. The results of these are given in Table 2.

TABLE 2: RESULTS OF TESTS ON PAIRED SERA FROM PATIENTS
I. THE ACUTE OR EARLY CONVALESCENT PHASE
II. THE LATE CONVALESCENT PHASE

No.	*Patient's Initials*		*Challenge R.O. Strain of Virus Dilution of Virus Suspension*			
			1:10	1:100	1:1,000	1:10,000
1	L.B.	I	+			
		II	−	+		
2	G.B.	I	−			
		II	+			
3	G.S.	I	+			
		II	+			
4	B.W.	I	−			+
		II	−	−		+
5	K.E.	I	−			
		II	−			−
6	R.G.	I	−	−		
		II	−	+		
7	J.M.	I	+			
		II	−	+		
8	A.S.	I	−			−
		II	−			+
9	C.S.	I	−	−	−	+
		II	−	−	−	+
10	L.W.	I	−	−	−	−
		II	−	+		
11	E.W.	I	−			
		II	+			
12	G.B.	I	−			
		II	+			
13	J.C.	I	−	−		−
		II				−
14	W.R.	I	−			
		II	+			

COMMENT

Of 14 patients, seven showed a change from negative protection test to positive protection when the acute phase serum was compared with the convalescent phase serum.

Of the remaining seven patients two gave negative results and five gave positive results in both tests. As the first blood may have been collected from the latter group in the early convalescent phase, it must be assumed that they had already developed antibodies as a result of infection. It thus appears that in a significant proportion of the cases antibodies to the virus developed during, and presumably as a result of, the infection. It is concluded then that this virus was responsible for this outbreak of Bornholm disease.

DISCUSSION

On the clinical findings a diagnosis of Bornholm disease was made. This is defined as an acute infectious illness characterized by a sudden onset of upper abdominal or lower thoracic pain, frontal headache and fever lasting a few days with a tendency to relapse once or twice.

This clinical syndrome was first observed in 1856 in Iceland by Finsen who called it pleurodynia. Since then epidemics have been recognized in all parts of the world. Many have been recorded in Scandinavia. In a classic monograph on the subject Sylvest[1] gave this condition the name Bornholm disease after the Danish Island of Bornholm in the Baltic. The same disease has been called various other names. Some are based on the geographical site of its occurrence, such as Skien disease, Bamle disease, Drangedal disease. Others are based on the most prominent clinical symptoms, such as epidemic diaphragmatic spasm, epidemic benign pleurisy, and devil's grip.

The disease occurs in epidemics usually in the summer and autumn. The incidence of infection is usually greatest in children and young adults. The incubation period is from 2–4 days. The onset is sudden, usually with pain

in the region of the diaphragm, which may be very severe and associated with protective splinting of the lower chest and upper abdomen. It is aggravated by coughing, sneezing, laughing and even breathing. Pain and tenderness of the muscles of the trunk and limbs may also occur. The patient develops fever, a rapid pulse, generalized aches and frontal headache, sometimes signs of meningeal irritation and occasionally meningo-encephalitis.

In adult cases, pleural involvement often occurs and a coarse pleural friction rub may be detected. A few cases also develop signs of pericarditis and more rarely of orchitis.

Numerous attempts to isolate the causative organism have been made. Bacteriological studies have given negative results. Many attempts to isolate a virus also failed until 1949, when Curnen, Shaw and Melnick [2] isolated a virus of the Coxsackie group from a boy with symptoms of pleurodynia. They [3] also noted that in 6 laboratory workers who accidentally contracted infection with Coxsackie viruses, 4 had illnesses resembling Bornholm disease. Weller, Enders, Buckingham and Finn [4] then reinvestigated specimens collected from patients who had pleurodynia in the 1947 epidemic in Boston. Strains of virus pathogenic for infant mice (the hallmark of a Coxsackie virus) were recovered from the throat washings of 4 patients. An increase in the titre of neutralizing antibody against this strain was detected in the sera of 7 patients collected in convalescence compared with the serum collected in the acute phase of the illness. Thus evidence was provided that pleurodynia was related to an infection with a strain later identified as belonging to the B group of the Coxsackie viruses. Since then considerable further evidence has accumulated indicating that Bornholm disease may be caused by an infection with Coxsackie virus, particularly the group B strains. It is therefore of significance that a virus having the characteristic of B group of Coxsackie or C viruses was isolated from the faeces of a significant proportion of the patients in this outbreak at Middelburg.

The criteria which distinguish the Coxsackie group from other viruses are their capacity for producing clinically manifest disease in infant mice and their relative avirulence for adult mice. Based on the organs affected and the type of lesion produced, Dalldorf,[5] who first identified this group of viruses, classified them into 2 groups A and B. Group A includes at least 12 antigenically distinct types of virus, all of which cause similar symptoms and lesions in mice. The infant mice inoculated with types of this group develop flaccid paralysis of one or more limbs and generally die within 48 hours of the first sign of weakness. Microscopic examination of the tissues shows diffuse extensive hyaline degeneration and repair of most or all skeletal muscles. No lesions are seen in the central nervous system or other organs.

Infant mice infected with group B strains, of which 4 antigenic types have been differentiated, clinically have generalized spasms and tremors as well as weakness and paralysis. Histological sections show focal not generalized diffuse muscle lesions, such as are seen in mice infected with group A strains. A characteristic inflammation is found in the fat pads, best seen in the interscapular fat pad, which shows areas of necrosis and an inflammatory reaction consisting of the infiltration of leucocytes and monocytes.[6]

Some of the types of the B group, including the strain isolated in this study, are also pathogenic to adult mice and produce characteristic lesions in the pancreas. These pancreatic lesions were first noted by Pappenheimer and others who described them in adult mice inoculated with the Conn 5 strain, later identified as Coxsackie group B type 1 virus.

Immunological studies of the relationships of the virus isolated from the Middelburg patients have revealed that it is a Coxsackie group B type 4 virus. This strain, in addition to being pathogenic and causing characteristic lesions in baby mice is also pathogenic to adult mice, causing an extensive inflammation and destruction of the pancreas. This finding suggests the possibility of inflammation of the pancreas in human patients. In many of them it is conceivable that the acute characteristic pain across the upper part of the abdomen may have been pancreatic in origin. This question could be settled by biochemical studies of pancreatic function in such patients, but this was not done in the present outbreak. The answer must await further investigations in other outbreaks.

SUMMARY

In January and February, the late summer of 1952, an epidemic of an illness characterized by signs and symptoms of Bornholm disease or pleurodynia affected the children of a school hostel in Middelburg, Transvaal.

The illness was sudden in onset in most cases with acute epigastric pain and tenderness often associated with severe headache, nausea, vomiting and fever. The fever lasted 1–3 days. In one third of the cases the illness relapsed with a recrudescence of the signs and symptoms, and in one tenth of the cases a second relapse occurred. In children the abdominal signs and symptoms were most obvious. In 5 adult cases chest signs and symptoms were more prominent.

In a laboratory study it was shown that this illness was associated with an infection of a Coxsackie group B type 4 virus. In addition to being pathogenic to and producing characteristic lesions in baby mice, this virus was also pathogenic to adult mice, causing in some an extensive destruction and inflammation of the pancreas. The possibility of involvement of the pancreas in human cases is mooted, but no studies of this possibility were made in this outbreak. It is now becoming clear that outbreaks of Bornholm disease are caused by infection with Coxsackie virus, particularly by strains of the B group. This view has received further support from the results of this study.

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DIE POST-OPERATIEWE RADIOLOGIESE VERSKYNSELS IN 'N GEVAL VAN PROLAPS VAN MAAGSLYMVLIES IN DIE DUODENUM

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Die post-operatiewe radiologiese verskynsels in 'n geval van sirkulêre of buisvormige prolaps van maagslymvlies in die duodenum, het nou bekend geword. Die geval, A. K., 'n 50-jarige man, is voorheen in hierdie *Tydskrif* beskrywe.[1]

Dit is miskien van waarde om die gegewens te publiseer omdat, eerstens, sover ons bekend opnames van hierdie aard nog nie vantevore die lig gesien het nie. Omdat prolaps van maagslymvlies in die duodenum gewoonlik gepaard gaan met ander afwykinge, waarvan die belangrikste maagulkus, duodenaalulkus en pylorusstenose is, is die operatiewe behandeling gewoonlik gedeeltelike maagreseksie. In hierdie geval, egter, is daar alleen gastrotomie verrig waardeur die oortollige maagslymvlies gemobiliseer en verwyder is. Na sluiting van die insisie is die maag en duodenum verder onaangeraak gelaat. Die geval bied dus 'n geleentheid om die radiologiese bevindinge van voor en na die operasie met mekaar te vergelyk.

Tweedens is daar ook, soos reeds aangetoon, onsekerheid oor die kliniese en ander verskynsels waartoe die kondisie mag aanleiding gee. Hierdie aspek geniet aandag en is onlangs weer elders bespreek.[3]

Die pasiënt is tweekeer radiologies ondersoek, 18 maande na die operasie. Daar was 'n tussenpose van 14 dae tussen die ondersoekings. Die bevindinge was in albei gevalle essensieël dieselfde, maar omdat die fluoroskopiese beeld by die eerste geleentheid nie so bevredigend was nie, weens tegniese faktore, word die tweede ondersoek hier beskrywe. Die beskrywing word gebaseer op die moderne opvatting van die anatomie en fisiologie van die pyloroduodenale oorgang, soos voorheen verduidelik.[2]

Radiologiese Verskynsels (8 November 1952). Alle opnames word in dieselfde posisie gemaak, nl. met die pasiënt staande in die regs-voor-skuinse houding (R.V.S.). Die maag het 'n normale haakvorm en is normaal van tonus. Die prepyloriese slymvlies is growwer as normaal. Peristaltiek verloop normaal tot by die prepyloriese gebied, wat effens spasties is maar by sommige geleenthede tog goed ontspan en saamtrek. Ontlediging normaal.

Terwyl die prepyloriese gebied vry is van peristaltiek (ontspanning van die canalis egestorius), is die bulbus volkome normaal (Fig. 2). As 'n peristaltiese golf tot teenaan die pyloriese opening verloop verskyn daar 'n klein vullingsdefek in die basis van die bulbus aan die groot kurvatuur kant. Hierdie vullingsdefek bereik sy maksimale grootte met maksimale kontraksie van die prepyloriese gebied, m.a.w. wanneer die canalis egestorius saamgetrek is en die pyloruskanaal ten volle gevorm is (vorheen ook systole van die antrum genoem) (Fig. 3). Die sambreel- of paddastoelvormige bulbus van voor die operasie (Fig. 1) is nie weer gesien nie, nóg gedurende deurligting, nóg op een van die opnames. Altesaam is 26 bulbus opnames gemaak vir argief doeleindes.

Kliniese Verskynsels. Die aanvalle van krampende pyn in die epigastrium, waarvan die pasiënt pre-operatief soveel las gehad het, het 18 maande na die operasie nog nie weer voorgekom nie. Die eetlus is goed en hy kan alle voedsel verdra.

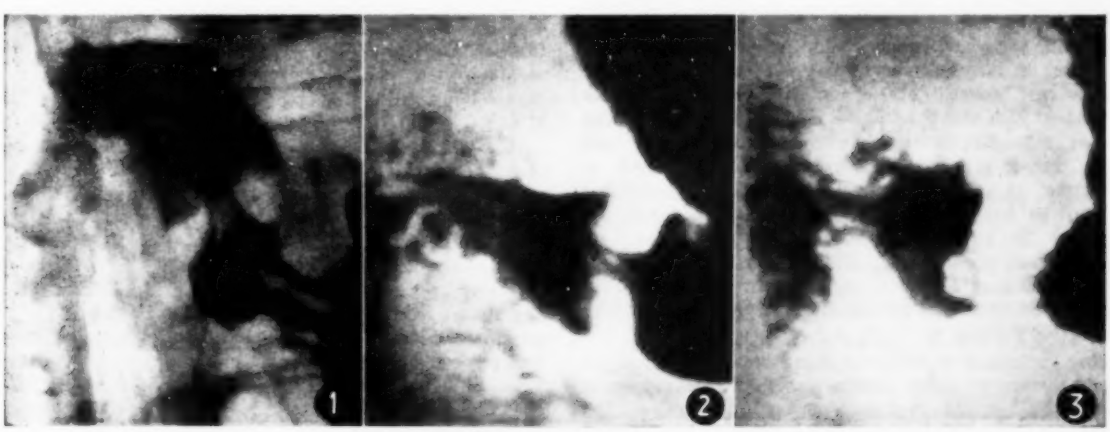

Fig. 1. Pre-operatief. Paddastoelvormige bulbus duodeni. Die canalis egestorius is maksimaal saamgetrek.
Fig. 2. Post-operatief. Normale bulbus. Die canalis egestorius is ontspan.
Fig. 3. Post-operatief. Vullingsdefek in die basis van die bulbus. Die canalis egestorius is ten volle saamgetrek.

GEVOLGTREKKING

Eerstens kan uit hierdie geval afgelei word dat daar na die operasie nog 'n sekere graad van prolaps van maagslymvlies in die duodenum aanwesig is, hoewel veel minder as daarvoor. Dit word aangedui deur die aanwesigheid van 'n klein vullingsdefek in die basis van die bulbus. Dit is te verwagte dat by 'n operasie van hierdie aard dit moeilik is om presies te besluit hoeveel slymvlies verwyder moet word.

Tweedens word afgelei dat die paddastoelvormige bulbus inderdaad deur prolaberende maagslymvlies veroorsaak is, omdat die beeld opgehewe is na verwydering van die grootste gedeelte van die oortollige slymvlies.

Derdens is dit weereens duidelik dat die verskyning van die vullingsdefek in die bulbus afhanklik is van die graad van kontraksie van die canalis egestorius, en nie van die posisie van die pasiënt en ander faktore nie, soos so dikwels beweer is.

Vierdens kan afgelei word dat die simptome deur hierdie bepaalde anatomiese afwyking veroorsaak is.

SUMMARY

The post-operative radiological features in a case of circumferential prolapse of gastric mucosa into the duodenum are here described for the first time, as far as we are aware. During 2 examinations, carried out 18 months after the operation, the bulb never exhibited the umbrella-like appearance seen pre-operatively; in the majority of views the bulb appeared quite normal, in some there was a small filling defect on the greater curvature side of the base. This indicates that a certain degree of prolapse is still present, but that the volume of prolapsing mucosa has been greatly reduced. The filling defect only appeared during contraction of the 'canalis egestorius' or, in other words, during antral systole.

The symptoms have not recurred.

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TWO NEW SALMONELLA SPECIES ISOLATED IN NORTHERN RHODESIA

SALM. NCHANGA AND SALM. CHINGOLA

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During the routine examination of food handlers, a non-lactose fermenting organism was isolated from the faeces of an African male, aged 23 years. This patient felt perfectly well and gave no history of recent illness, although he was found to be a carrier of amoebic dysentery.

The non-lactose forming organism was not present in the faeces in large numbers as it was isolated only after selenite enrichment. This gram-negative motile bacillus fermented, with the production of acid and gas, glucose, maltose, mannitol, dulcitol, sorbitol, arabinose, rhamnose, xylose, trehalose, inositol; dextrin was fermented with the production of acid and gas, after some days' incubation. It failed to ferment lactose, salicin, adonitol, inulin and raffinose. The citrate utilization test was positive, H_2S was produced but urease and indol were not formed. The Vosges-Proskauer test was negative, the methyl red test positive. Gelatine was not liquefied after 22 days' incubation at 37° C.

When examined serologically, this organism was agglutinated to titre by *Salm. london* O serum. Absorption of this serum by the organism completely removed the homologous agglutinins; similarly, *Salm. london* was agglutinated to titre and absorbed all homologous agglutinins from a serum made with the organism. Therefore, the somatic structure is 111.X.

The culture was plated and single colonies tested: some were agglutinated by *Salm. london* H (l, v) serum and some by *Salm. newport* var *puerto rico* H (1, 2, 3) serum. When tested with absorbed single-factor sera, the former colonies were agglutinated by pure v serum, the latter by pure 2 and 3 sera. By absorption, it was found that a suspension of the new strain with the flagellar phase 1, v reduced the homologous titre of *Salm. london* H (1, v) serum from 1 : 12,800 to 1 : 400, but was able to remove completely all the 1, v agglutinins from *Salm. manchester* H (1, v) serum.

A suspension of *Salm. london* or of *Salm. manchester* removed all H (1, v) agglutinins from a serum made with the new strain. These findings show that the 1, v phase of the new strain is identical with that of *Salm. manchester*, but that *Salm. london* H (1, v) contains a minor additional antigen. The flagellar phase H (1, 2, 3) of the new type was agglutinated to titre and absorbed all homologous agglutinins from *Salm. newport* var *puerto rico* H (1, 2, 3) serum. Similarly, *Salm. newport* was agglutinated to titre and removed all homologous agglutinins from the serum made with the new strain. Therefore, the flagellar antigens are 1, v–1, 2, 3.

Summary. A new Salmonella type has been isolated from the faeces of a healthy African food handler. The organism has the structure 111.X., 1, v–1, 2, 3, and has been named *Salm. nchanga.*

SALM. CHINGOLA

A specimen of faeces was obtained from an African woman suffering from gastro-enteritis. Direct plating on desoxycholate citrate agar showed numerous non-lactose fermenting colonies. The colonies were typical of those of the Salmonella group. One colony was seeded and studied in detail. The organism was a gram-negative motile bacillus which fermented with the production of acid and gas, glucose, maltose, mannitol, sorbitol, arabinose, xylose, dulcitol, trehalose and dextrin. It failed to ferment lactose, sucrose, salicin, inulin, raffinose, adonitol and inositol. Indol and urease were not produced and gelatine was not liquefied after 24 days' incubation at 37° C. The Vosges-Proskauer test was negative, the methyl red test and citrate utilization test were positive.

The organism was agglutinated to titre by and absorbed all homologous O agglutinins from *Salm. aberdeen* O serum; similarly, *Salm. aberdeen* O was agglutinated to titre and absorbed all homologous O agglutinins from a serum made from the new strain. Therefore, the somatic antigen is represented by the symbol XI.

It was found that 2 colonies could be isolated from the original culture, one of which was agglutinated to titre by *Salm. cambridge* H (e, h) serum, the other was agglutinated to titre by *Salm. newport* var *puerto rico* H (1, 2, 3) serum. When tested with absorbed single-factor sera the first colony was agglutinated by pure h serum, the latter by pure 2 and pure 3 sera. It was found that phase 1 of the new organism completely absorbed all H (e, h) agglutinins from *Salm. cambridge* serum; similarly the *Salm. cambridge* removed all H (e, h) agglutinins from a serum made from the new species. In addition *Salm. newport* var *puerto rico* H (1, 2, 3) absorbed all H (1, 2, 3) from a serum made with the new strain which removed all homologous H agglutinins from *Salm. newport* var *puerto rico* H (1, 2, 3) serum. The flagellar antigens are therefore e, h–1, 2, 3.

Summary. A new Salmonella species has been isolated from the faeces of an African woman suffering from gastro-enteritis. This organism has the antigenic structure XI, e, h–1, 2, 3 and has been named *Salm. chingola.*

Acknowledgements and thanks are due to the Management of Nchanga Consolidated Copper Mines Limited, in whose hospital laboratory these organisms were first isolated, for permission to publish this paper. Thanks are also expressed to Dr. L. R. B. Birt, the Chief Medical Officer at the time, for his interest and co-operation in the further investigation work which was carried out.

HYPOTENSIVE ANAESTHESIA IN PLASTIC SURGERY

A REPORT ON 500 CASES

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and

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Following a previous paper on this subject published in this *Journal* a year ago, and reporting on 100 operations under hypotensive anaesthesia, we consider it advisable to submit a further report on a greater number of cases in order to encourage the use of this form of anaesthesia in other fields of surgery.

Method. There has been no fundamental change in the method used. Induction is carried out with Intraval sodium 0.5 gm. mixed with Flaxedil 60–80 mg. Intubation is essential in all cases under hypotensive anaesthesia to make certain of a perfect airway. After packing off the throat and settling with gas, oxygen and ether, maintenance with 100% oxygen and ether is the rule. There is no objection to maintenance with gas and oxygen, plus small doses of Pethidene or Intraval, or gas, oxygen and Trilene. These alternatives are used when the patient seems to resent ether, or when the surgeon wishes to use diathermy. The supply of 100% oxygen and the smooth and sufficient anaesthesia afforded by ether are additional safety factors in the successful production of hypotension.

Although some writers have objected to ether on the grounds of post-operative vomiting and delayed post-operative recovery, these complications have not been prominent in this series. Vomiting occurred in less than 10% of cases, possibly because Hexamethonium retards the motility and secretomotor activity of the stomach.[1] So far as post-operative recovery is concerned, experience teaches that very little ether is required once hypotension has been produced. The average post-operative recovery period was 10–15 minutes after completion of the operation. This interval is useful in that it allows time to settle the patient in bed in the desired position before consciousness is regained, and although some patients do take up to 30 minutes to recover consciousness, this is considered to be due to the hypotension rather than to the ether. It is also noteworthy that less Hexamethonium is required to produce the desired hypotension with ether than with the other agents.

After settling the patient the table is tilted into a reverse Trendelenburg position of 10–15° in older patients, and up to 35° in younger ones. A Gordh needle is then inserted in any suitable and convenient vein and the Hexamethonium injected. In this series only Hexamethonium bromide was used. The dose varied from 40–80 mg. in the young, healthy adult, to 10–25 mg. in the elderly or hypertensive patient. At first it was found that the blood pressure of young, healthy adults had a tendency to come down to a suitable level and then to rise sud-

denly to almost normal, despite the addition of big doses of Hexamethonium. A modification, since introduced, is the addition of 2–3 c.c. of Procardyl 10% (procaine amide hydrochloride M & B) immediately after injection of the Hexamethonium.[2] It is well known that procaine amide itself causes a hypotension if injected rapidly under anaesthesia. If injected slowly, it does not cause a further fall in blood pressure, but appears to stabilize the hypotension produced by the Hexamethonium. Furthermore, the weak link in the hypotensive chain is the myocardium,[3] and it may well be that Procardyl is an added safety factor as one of its main uses in medicine is to restore cardiac regularity.

One tries to control the hypotension by varying (a) the dosage and (b) the angle of tilt. Control is essential in all cases above the age of 40 and in hypertensive patients. When the patient is a healthy, young adult and the pressure drops slightly below the desired level after the initial doses of Hexamethonium and Procardyl, in order not to inconvenience the surgeon unduly by frequent alteration of the tilt, and while awaiting the return of the blood pressure to the desired level, reliance is placed on the following signs which, incidentally, are of value in all cases:

1. *Breathing.* If the breathing is regular with no tracheal tug, all is well as a rule. If the breathing is irregular or if a tracheal tug is present, cerebral anoxia may be responsible and the pressure should be raised.[4] These signs occur also in deep anaesthesia, but anaesthesia in plastic surgery is sufficiently light to exclude this possibility.
2. *The Skin.* The skin should be pink and warm. If mottled and cold the pressure should be raised.
3. *Operation Site.* With adequate hypotension, anaesthesia and oxygenation, the operative field should be comparatively dry with little capillary bleeding, and the colour of the blood should be pink. With inadequate hypotension and oxygenation, and where hypotension is produced without tilting, there may be troublesome venous oozing and cyanosis.[5]
4. *Peripheral Circulation.* The lobes of the ears, the finger nails and the toe nails should be pink and warm at all times with normal filling of the capillaries on pressure.

On completion of the operation the patient is tilted back to the horizontal position or with the head down until the required pressure (70 mm. Hg in young adults, and 80 mm. Hg or upwards in hypertensives or elderly patients) is obtained. He is then taken back to the ward and placed in the desired position. Moving should be gentle because rough handling leads to wide variations of pressure. Oxygen is given and a trained nurse is present until the patient recovers consciousness. If the pressure does not return to the desired level in the usual time (15–20 minutes in hypertensives and older patients, and 30–40 minutes in healthy, young adults), Methedrine should be used in graduated doses to restore it, i.e. 2–3–5–10 mg. It is not necessary to inject larger doses of Methedrine, which might conceivably cause a reactionary haemorrhage in the operation field. Small repeated doses with continuous blood pressure readings will raise the level of the pressure adequately; 7% of cases in this series received Methedrine.

Results. To date 500 cases have been treated. The results were:

456 cases: Good (approximately 91%).
35 cases: Fair (7%).
9 cases: Failures (approximately 2%).

Ages. The youngest patient was 14 and the oldest 78.

Complications. One post-operative deep vein thrombosis occurred in the left calf of a stout woman aged 38, after a nasal operation. This may have been due to inadequate padding of the foot-piece at the time, but possibly the state of hypotension was an aggravating factor.

REMARKS

1. The vast majority of these operations was carried out in one nursing home with a trained staff and with facilities which allowed the anaesthetist to be within a very short distance from the wards. We consider this factor, and the team-work between anaesthetist, surgeon and the nursing staff, to be essential for the safety and success of the anaesthetic.

2. Other papers on hypotensive anaesthesia state that a systolic blood pressure of 70 mm. Hg is adequate from the point of view of the surgeon. This was too high for practical purposes in this series of cases, possibly due to the altitude of this city (5,800 feet). Under Hexamethonium, the optimum blood pressure level in Johannesburg was found to be approximately 60 mm. Hg in hypertensives and older patients, and 50 mm. Hg in young, healthy adults. It is interesting to note that the failures occurred in patients who had come up from sea level the day before operation.

3. The method is used only on patients regarded as being normal anaesthetic risks.

4. So far as possible controlled hypotension was not used for longer than 1½ hours. When an operation required longer anaesthesia, controlled hypotension was utilized only while the essential stage in that operation was being carried out, after which the pressure was allowed to rise to 80–90 mm. Hg with the knowledge and consent of the surgeon. A slow blood transfusion drip was of considerable value in these cases.

CONCLUSION

Almost every type of plastic operation 'above the belt' has been performed under the system of anaesthesia indicated in this paper. There is no doubt that operating under such a system ensured an even, rapid technique, reduced trauma and afforded a smoother convalescence with a minimum of oedema and pain in the operation field.

So long as patients are carefully selected, adequate anaesthesia with full oxygenation guaranteed, and intelligent co-operation between surgeon, anaesthetist and nursing staff exists, the introduction of hypotensive anaesthesia in plastic surgery is without doubt a most important advance.

Our thanks are due to Dr. G. E. Hale-Enderby for his helpful interest and suggestions at all times, and also to May & Baker for making 'Procardyl' available for our use.

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ACTH AND EPILEPTIFORM SEIZURES IN CHILDHOOD

A PRELIMINARY REPORT

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It is well established in clinical practice that some forms of epilepsy are very difficult to control with all the well-known anti-convulsant drugs. Many of the uncontrollable cases are those of children where, indeed, the problem of adequate and immediate treatment is urgent because of the slow mental deterioration that may even tuate.

In the Department of Neurosurgery of the Johannesburg General Hospital we encounter cases in children with great frequency. The children suffer from *status petit mal* which does not respond to either Tridione or Paradione in quite large dosages, even though these drugs are coupled with others such as Phenobarbitone and Epanutin.

It is suggested as a possibility that these cases which exhibit attacks resembling *petit mal*, sometimes so frequent as to produce clouding of consciousness over a long period and accompanied by imbalance of gait, may be caused by scattered bilateral areas of demyelination. This demyelination may in its turn be the result of previous viral infection, perhaps subclinical and followed by an auto-antigen-antibody sensitization reaction. It was on such a purely theoretical basis that ACTH was first given as an empirical measure. On the other hand, ACTH may produce its effect by its action in changing the biochemistry of the internal environment, and thereby acting on the presumed epileptogenic foci of the diencephalon. As ACTH does on occasion favourably influence *status petit mal*, where no previous history of encephalitis exists and where investigations, apart from the EEG, are all negative, the latter explanation may be more accurate.

We are at present investigating a large series of cases of convulsive disorders in childhood in relation to their response to ACTH. We take this opportunity of reporting on our first 2 cases employing ACTH as a therapeutic agent.

CASE NO. 1

G.M., age 3, male, was seen for the first time by us on 28 February 1951.

In January 1950 this child commenced to have blackouts. On the first occasion he was found on the floor of his room quite unconscious. His eyes were open, but he lay quite limp. One month later he had a similar attack and then 2 weeks later he had the first attack which was witnessed. There was jerking up of both arms and he fell over backwards. The attacks have increased in frequency since that time. He got convulsive movements on both sides of his body, went pale and slightly cyanotic, and had marked movements of his mouth which were jactitations. He never frothed at the mouth nor bit his tongue, but was incontinent. The attacks lasted 5 to 10 minutes.

At the onset of his blackouts he had no serious illnesses and no temperature. His birth was normal and he was quite healthy in infancy, reaching the normal landmarks of development at the normal times. There was no history of epilepsy in the family.

In March 1950 he had tonsillitis and bronchitis. In June 1950 he had an enteritis with a temperature of 105° F.

His attacks since January 1950 have varied from the severe form described above, to slight minor types indistinguishable from *petit mal* seizures.

At the time of our first consultation, he was eating and sleeping well, and his bowels were regular.

On Examination: The child appeared to be in fairly good health apart from the convulsive episodes. After bouts of minor attacks he was very confused. He did not know his whereabouts, or recognize his parents, and his speech was incoherent and indistinct. There was no papilloedema and no field defect. No abnormalities in the sensory and motor fields were observed.

He was admitted to our wards in the Department of Neurosurgery on 8 April 1951.

X-ray Skull: No abnormalities were revealed.

Lumbar Puncture: The fluid was clear; no block was present. Pressures were difficult to obtain. No cells were observed. Total protein was 10 mg. per 100 c.c. The Eagle flocculation test was negative.

Air Encephalogram: This revealed a mildly abnormal filling, without shift of the ventricular system, but with slight generalized dilatation consistent with some degree of atrophy.

White Cell Count: 8,800 leucocytes per c. mm.; neutrophils, 66%; eosinophils, 1%; monocytes, 1%; lymphocytes, 1%.

No immature or atypical cells were observed.

EEG (a day before air encephalography): 'Recorded simultaneously on 8 channels using 16 scalp and 2 ear leads.

In unipolar leads the dominant rhythm was a high voltage (up to 150 μv activity at about 5–6 per second with some overlying faster activity. This theta activity was more marked in the parietoocipital regions. The occipital leads showed some asymmetry, with slower activity at a higher voltage on the left side.

Among this theta rhythm there were also some high voltage delta waves, with flat or notched tops, at 3–4 per second.

In bipolar leads there was a lot of out of phase activity,

particularly over both parietal regions. No wave and spike pattern was seen.

Conclusion: This was an abnormal record, and suggested scattered areas of abnormal brain activity.'

Glucose Tolerance Test: Normal.

The final diagnosis was presumed to be a leucoencephalopathy with epileptiform phenomena. Attacks continued while in hospital. Tridione gr. 4½ four times per day by mouth with Phenobarbitone gr. ½ t.d.s. did not result in improvement. Similarly, Paradione and Phenobarbitone were of no avail.

Finally, the child was given ACTH 10 mg. three times per day, from 15 April 1951. A dramatic response was noted within 3 days. Whereas fits had been frequent daily, they suddenly abated. The improvement continued for a further fortnight. The dose of ACTH was reduced to 10 mg. b.d. for a further week, and the child still remained free of attacks. He was discharged home on a maintenance dose of 10 mg. daily.

In July a letter from his father stated that the boy remained well. A further course of ACTH 10 mg. three times per day was given for 3 weeks. His improvement was maintained. He remains well until the present, apart from occasional minor attacks.

CASE NO. 2

P. S., age 3½ years, female, was referred to us on 5 July 1951, with a history of convulsions since December 1950.

Family History: The parents have been separated recently. The child has been living with her father and has apparently been looked after fairly well. The parents are both healthy. One elder step-sister, aged 14 years, is healthy.

Past History: Nil significant.

Habits: Bowels and bladder well controlled.

History: Convulsions first occurred in December 1950, the child being quite well previously. The convulsion was described by the parents as having no localizing features and was in the nature of a tonic and clonic fit. It was a very violent convulsion and lasted about 5 minutes. Her temperature rose to 105° F following this. The next day she was quite well. Another fit occurred one week later, similar to the one described above. The attacks were recurrent after this. They occurred at a rhythmical interval of one week out of 4 every month. They showed no localizing features. Some were minor attacks, with just slight facial twitching and lapse of consciousness lasting a few seconds, or there were occasional *grand mal* attacks. They sometimes went on for 24 hours. During this period the child was mentally confused and unable to hold herself up straight. Occasionally she foamed at the mouth. She was investigated in the Children's Hospital in January 1951. X-ray of the skull showed no abnormalities. An air encephalogram was done at the Children's Hospital and this was essentially normal. The EEG showed an epileptic pattern.

On Examination: The child appeared mentally confused. She would not answer questions. She stared, with her mouth wide open. Neurologically, there was nothing abnormal. Repeat EEG's showed bilateral large voltage slow waves in all leads, suggestive of idiopathic epilepsy.

Lumbar Puncture: Pressure 110, fluid clear, no block.

Cerebrospinal Fluid: Cell Count: 1 lymphocyte per c.mm. Total Protein: 30 mg. per 100 c.c.

This child continued to have major convulsions and was very confused. A diagnosis of severe idiopathic epilepsy with *status petit mal* was made.

A series of fits began on 20 July. There was a space of 2 days. On 23 July she had a fit. On the 24th, a fit; on the 25th, 2 fits; on the 26th, 3 fits; the 27th, 1 fit; the 28th, 3 fits. The fits were *grand mal* in type. From 20-28 July her mental condition indicated considerable confusion. She lay in bed and stared absently into space. She had to be fed and was incontinent. She did not respond to questioning and her voice was slurred. It was decided to give her ACTH.

28 July 1951. A course of ACTH was commenced. 10 mg. 6-hourly on the first day.

29 July 1951. 5 mg. ACTH 6-hourly. For 24 hours after the commencement of ACTH the patient had not had any fits. Her mental condition improved. She was alert and talkative.

2 August 1951. The EEG showed normal frequencies for her age. ACTH 5 mg. b.d.

5 August 1951. No fits since the ACTH. 5 gm. daily from to-day.

6 August 1951. Slight fit to-day. Mild twitches. She is still on ACTH.

11 August 1951. The child has had slight fits each day since the last report. It was decided to discontinue her ACTH.

18 August 1951. No fits since the last report. She remains bright and cheerful.

25 August 1951. She continues to remain well. She is taken out by her mother and father in the afternoons. Plans are being made to get her home.

3 September 1951. The patient had a fit yesterday, which left her confused. She is on Alepsal 1 capsule t.d.s. and Paradione 1 capsule t.d.s. Since 25 August she has been on Alepsal 1 capsule t.d.s. The Paradione is to be started to-day.

10 September 1951. Attacks have recurred. She has had an occasional epileptic fit but her mental condition is confused. She is dysarthric. Her general musculature is hypotonic. She lies in bed and barely takes notice of her surroundings. ACTH 5 mg. 6-hourly given.

13 September 1951. Her condition has improved. She is taking notice again.

18 September 1951. Her condition has returned to normal. In view of this the father is taking her out this week and is having a trained sister to take care of her. She is on Alepsal, 1 tablet t.d.s. ACTH has been stopped. The father has been told that if she deteriorates a biopsy will be taken.

The child relapsed into similar attacks in October 1951. She was admitted to the Florence Nightingale and put on ACTH 5 mg. 6-hourly. She improved again within a week. She was kept under observation until January 1952. ACTH was gradually cut down to 5 mg. daily. On 31 January she was discharged. She was given ACTH 5 mg. on alternate days at home. Although there was some increase in the frequency of her attacks for a while, she gradually improved. She was improving mentally and physically. On 3 June 1952 the ACTH was discon-

tinued and she was put on Phenobarbitone grain ¼ b.d. and Epanutin, grains 1½ *mane*. She has few attacks now and the parents are well satisfied with her improvement.

SUMMARY

1. Two cases of children presenting with frequent *petit mal* attacks which proved resistant to well-known anti-convulsant drugs, were put on ACTH, 5 mg. 6-hourly, or 10 mg. 3 times per day, gradually reducing the dosage.

2. Improvement was dramatic and maintained, the children remaining well until the present.

3. ACTH is presumed to work on the basis of an auto-antigen-antibody reaction in diseased brain tissue, or else as directly affecting epileptogenic foci, biochemically.

NEW PREPARATIONS AND APPLIANCES

CHEMICAL STRUCTURE AND GENERIC NAME FOR TERRAMYCIN—PFIZER

The chemical formula of the antibiotic sold under the trade-mark Terramycin was recently announced in the *Journal of the American Chemical Society* (74 : 3708 : 20 July 1952). As a result of the discovery of its chemical structure, the name 'oxytetracycline' has been adopted as the generic name of this antibiotic. Its molecule contains a high proportion of oxygen, which is believed to account for the compatibility of the antibiotic with body fluids and tissues. Further, its amphoteric properties permit the formation of both acid and basic salts, assuring full availability through the pH range of the gastro-intestinal tract. Absorption is both dependable and rapid.

The World Health Organization has now accepted for general use the generic name 'oxytetracycline' and it is anticipated that, in the near future, the literature will contain numerous references to the generic name.

Arrangements are being made for all Terramycin products to be labelled with both the generic and proprietary names.

IN MEMORIAM

DR. J. F. HAEGERT

After a long and trying illness lasting for more than 2 years, Dr. J. F. Haegert died in Ladybrand on 30 March 1953.

Dr. Haegert was born in Calcutta in 1878, where his father was a missionary doctor. At the age of 24 he received his M.B., Ch.B. degree from the University of Edinburgh, and after working in various hospitals in the British Isles he returned to India, working his passage over as ship's surgeon. From 1906 to 1911 he practised in Patagonia. Life there was primitive and dangerous, and he relished the memories of his experiences there. In 1911 he returned to Edinburgh and received his M.D. degree.

He then came to South Africa and settled in Ladybrand where he practised until illness overtook him.

Dr. Haegert was a lover and keen observer of Nature, and published a number of articles on animals and birds. He was well known to the older generation of the profession, who would remember him for various medical contributions to this *Journal*. He loved his work and was an astute diagnostician, relying on his clinical skill rather than upon the modern diagnostic aids.

His last years were extremely trying after he had lost the use of his limbs and eyes. Some of his old patients eased his sufferings by reading to him. His deep religious sense did not allow for complaints.

He leaves a wife, a son and 2 daughters.

PASSING EVENTS

Dr. Louis F. Freed, of Johannesburg, is one of the authorities quoted in the 'Report on Divorces in Johannesburg', published in 1951 by the Social Welfare Department of the City of Johannesburg. Dr. Freed's article, 'The Psychic Changes Projected by Disorganization of the Central Nervous System', which appeared in the *South African Medical Journal* in 1951, has recently attracted the attention of Professor Carlo Berlucchi, Direttore, Clinica malattie nervose e mentali della Universita Pavia.

UNIVERSITY OF THE WITWATERSRAND APPEAL

The £1,000,000 Appeal of the University of the Witwatersrand is being launched by the Mayor of Johannesburg, Mr. H. Miller, on Monday 11 May 1953, when he will open the Exhibition to be held at the University, Milner Park, Johannesburg. The Exhibition will be on view for the week 11–16 May from 2 to 10 p.m. and on Saturday 16 May from 10 a.m. to 10 p.m. There will be a central exhibit in the foyer and examination hall, while the various departments and laboratories will be on display. The Medical School is having a special exhibit in the Pathology Museum, where as many departments as possible will take part. Where practicable, the non-clinical laboratories will be available for public inspection. At the same time continuous film-shows on medical topics of public interest will be held in the Harveian Lecture Theatre. The public and medical profession are cordially invited to attend any of the exhibits or departments in which they might be interested.

COLLEGE OF GENERAL PRACTITIONERS, LONDON

The first meeting of the fully constituted Foundation Council of the College of General Practitioners was held recently in London. At this meeting the Provisional Bye-Laws were amended to the effect that Membership and Associateship will not be confined to persons with qualifications registered with the General Medical Council but is extended to any general practitioner with equivalent registration in a country or state within the British Commonwealth or the Republic of Eire, who may join the College without further formalities.

The address of the College is 14 Black Friars Lane, Queen Victoria Street, London, E.C.4, England.

INTERNATIONAL MEDICAL CONGRESSES

The following list of forthcoming congresses has come to hand:

Date	*Place*	*Name*	*Office*
21–26 April	Rotterdam	General Assembly of the International Union against the Venereal Diseases.	Secretariat: Dr. Cavaillon, Institut Alfred-Fournier, 25, Bd. Saint Jaques, Paris-14e, France.
2– 6 May	Bologna	18th Int. Congress of Otoneuro-ophthalmology.	Secretary-Gen.: Dr. G. Cristini, Clinica oculistica Policlinico, Bologna, Italy.
4– 9 May	Paris	Congress of the Int. Bureau of Epizootics.	Secretary-Gen.: Prof. Ramon, Office int. des epizooties 12, rue de Prony, Paris-17e, France.
14–16 May	Brussels	Congress of French Speaking Paediatricians	Information: Prof. R. Dubois, Clinique de médecine infantile, 329, rue Haute, Brussels, Belgium.
20–23 May	Stockholm	European Congress of Allergology.	Secretary-Gen.: Dr. Egon Bruun, Gersonsvej 8, Hellerup, Copenhagen, Denmark.
25–30 May	London	8th Congress of the Int. Hospital Federation.	Secretary of the Federation: Captain J. E. Stone, King Edward's Hospital Fund for London, 10 Old Jewry, London E.C.2, England.
25–31 May	New York City	First World Congress on Fertility and Sterility.	Information: Dr. A. T. Weisman, 1160 Fifth Avenue, New York, U.S.A.
May	Paris	Int. Union for Public Health Education Plenary Meeting.	Information: Mr. L. Viborel, Centre int. de l'education sanitaire, 66, Bd. St. Michel Paris-6e, France.
5– 6 June	Groningen	Int. Congress of Audiology (preceded by a Course on Audiology, 1–4 June).	President: Dr. Gunnar Holmgren, Strandvagen 5A, Stockholm, Sweden.
6 June	Brussels	2nd Congress of the World Medical Press.	Secretary-Gen.: Dr. C. Mayer, 141, rue Belliard ,Brussels ,Belgium.
6– 7 June	Utrecht	Congress of the Int. Association for the Study of the Bronchi.	Secretary-Gen.: Dr. Swierenga, St-Antonius Hospital, Utrecht, Netherlands.
8–13 June	Amsterdam	5th Int. Congress of Otorhinolaryngology.	Secretary: Dr. W. H. Struben, J. J.
10–13 June	Rome	Congress of the Latin Society of Ophthalmology.	Secretary-Gen.: Prof. Filippi Gabardi, 34 via Torleone, Bologna, Italy.
13–17 July	Petropolis (Rio de Janeiro)	4th Congress of the Int. Council of Nurses.	Secretary: Miss Daisy C. Bridges, 19 Queen's Gate, London S.W.7, England.
19–25 July	Copenhagen	7th Int. Congress of Radiology.	Secretary-Gen.: Prof. F. Nordgaard, 100ster Voldgade, Copenhagen K, Denmark.
20–25 July	London	1st Int. Congress of Medical Librarianship.	Secretariat: London School of Hygiene and Tropical Medicine, Keppel Street, London W.C.1, England.
18–21 August	Boston	3rd Int. Congress of Electroencephalography and Clinical Neurophysiology.	Secretary-Gen.: Dr. Robert S. Schwab, Massachusetts General Hospital, Boston 14, Mass., U.S.A.
24–29 August	Geneva and Aix-les-Bains	8th Int. Congress of Rheumatic Diseases.	General Secretariat: Institut de physiatrie, Hôpital Cantonal, Geneva, Switzerland.
24–29 August	London	Int. Conference on Medical Education.	Organized by the World Medical Association. Secretary: Dr. Louis H. Bauer, 2 East 103rd St., New York, N.Y., U.S.A.
24–30 August	Bellagio, Italy	9th Int. Congress of Genetics.	Interim Committee: 10, via Celoria, Milan, Italy.
27 August–1 September	Paris	11th Int. Congress of Psychotechnology.	President: Prof. L. Bonnardel, 41, rue Gay-Lussac, Paris-5e, France.
28 August–4 September	Istanbul	5th Int. Congress of Tropical Medicine and Malaria.	Secretary-Gen.: Prof. Ihsan Sukru Aksel, Beyoglu Tunel, Meydani no.2, Istanbul, Turkey.
31 August–September	Amsterdam	Annual Meeting of the World Medical Assoc.	Secretary-Gen.: Dr. Louis H. Bauer, 2 East 103rd St., New York, N.Y., U.S.A.
31 August–4 September	Montreal	19th Int. Physiological Congress.	Secretary: Miss MacCallum, Donner Building, McGill University, Montreal, Canada.
1– 7 September	Milan-Stresa	Int. Congress of Logopedics and Phoniatrics.	Secretary-Gen.: Dr. Deso A. Weiss, 115 East 86th Street, New York 28, N.Y., U.S.A.
6–12 September	Rome	6th Int. Congress of Microbiology.	Information: Dr. V. Puntoni, President of the Italian Society for Microbiology, Città Universitaria, Rome, Italy.
7–12 September	Lisbon	5th Int. Neurological Congress.	Secretary-Gen.: Prof. A. Lima, Hospital Julio de Matos, 53, Avenida do Brasil, Lisbon, Portugal.
7–12 September	London	1st Congress of the World Confederation for Physical Therapy.	Hon. Secretary: Miss Neilson, c/o Chartered Society of Physiotherapy, Tavistock House (South), Tavistock Square, London W.C.1, England.
14–21 September	Lisbon	15th Congress of the Int. Society of Surgery.	Secretary of the Society: Dr. L. Dejardin, 141, rue Belliard, Brussels, Belgium.
September	New York and Montreal	6th Int. Congress of Medical and Surgical Film.	Information: Centre Int. du film médical et chirurgical, 3, rue de Siam, Paris-16e, France.
3–10 October	Madrid	6th Int. Leprosy Congress.	Secretary: Dr. Felix Contreras, Moreto 15, Madrid, Spain.
October	Italy, probably Rome	Congress of the Int. Union of Railway Medical Services.	Secretariat, for the time being, Medecin general Rouvillois, médecin conseil de la SNCF, 88, rue St-Lazare, Paris-9e, France.
December	Havana	7th Int. Congress of Paediatrics.	President of the Congress: Prof. Felix Hurtado, 5a Avenue, 124, Miramar, Havana, Cuba.

REVIEWS OF BOOKS

PSYCHIATRY

Practice of Psychiatry. By William S. Sadler, M.D., F.A.P.A. (Pp. 1183. £6 7s. 6d.) St. Louis: The C. V. Mosby Company.

Contents: 1. General Psychiatric Considerations. 2. The Pathoses. 3. The Neuroses. 4. The Psychoses. 5. Personality Disorders. 6. Psychosomatic Diseases. 7. General Psychotherapeutics. Appendix. Bibliography. Glossary.

The author explains that as psychiatric progress had been so marked since the publication of *Modern Psychiatry* 7 years ago, it was decided to re-write this book and call it *Practice of Psychiatry* rather than to attempt its revision.

One entirely new feature in this volume in the section entitled the *Attitudinal Pathoses*. This term embraces a group of disorders which are pre-neurotic, some of which are commonly referred to as 'maladjustments', 'complexes', etc. The use of this term to designate this new diagnostic category was suggested by Dr. F. C. Thorne. This is an extremely interesting approach and worthy of perusal.

This is indeed a comprehensive survey of the psychiatric field. All the sections and in particular those dealing with the psychosomatic diseases and psychotherapeutics are exhaustive and up to date. These features and the extensive bibliography make this volume a 'must' for all those concerned in the practice of psychiatry.

ROSE AND CARLESS' SURGERY

Rose and Carless' Manual of Surgery. Vol. 1 and Vol. 2. Edited by Sir Cecil Wakely, *Bt. K.B.E., C.B.* (Pp. 747–1471 with illustrations. 63s. for two volumes. Eighteenth Edition.) London: Baillière, Tindall & Cox.

Contents: *Vol.* 1. 1. The Pathogenesis of Infection. 2. Disorders of the Blood of Surgical Importance. 3. Blood Transfusion. 4. Haemorrhage and Shock. 5. Biopsy in Surgery. 6. Water and Salt Deficiency in Surgery. 7. Chemotherapy. 8. Specific Infective Diseases. 9. Wounds. 10. Wound Infections. 11. The Use of Physical Agencies in Surgery. 12. Injuries and Diseases of Arteries—Aneurysm—Ligature of Arteries. 13. Affections of the Veins—Angiomata. 14. Diseases of the Lymphatic System. 15. Affections of Nerves. 16. The Surgery of the Autonomic Nervous System. 17. Surgical Diseases of the Skin and Its Appendages. 18. Affections of Muscles, Tendons and Bursae. 19. Deformities—Orthopaedic Surgery. 20. Injuries of Bones—Fractures. 21. Diseases of Bone. 22. Injuries of Joints—Dislocations. 23. Diseases of Joints. 24. Injuries of the Spine. 25. Diseases of the Spine. 26. Affections of the Scalp and Cranium. 27. Affections of the Brain and its Membranes. 28. Affections of the Lips and Jaws. 29. Affections of the Mouth, Salivary Glands, and Palate.

Vol. 2. 30. Affections of the Nose, Paranasal Sinuses and Naso-Pharynx. 31. Affections of the Tonsils and Pharynx. 32. Affections of the Oesophagus. 33. Surgery of the Upper Air Passages. 34. Surgical Affections of the Lungs, Pleural Cavities and Mediastinum. 35. Surgery of the Neck. 36. Diseases of the Breast. 37. Abdominal Surgery. 38. Hernia. 39. Intestinal Obstruction. 40. Affections of the Rectum and Anus. 41. Surgical Affections of the Kidneys. 42. Bladder and Prostate. 43. Affections of the Testicle, Cord, Scrotum and Seminal Vesicles. 44. Affections of the Urethra and Penis. 45. Amputations. 46. Plastic Surgery. 47. Burns. 48. Surgery of the Female Genital Organs. 49. Surgical Affections of the Ear. 50. Surgical Affections of the Eye. 51. Tropical Surgery. 52. Anaesthesia. Index.

It is now 54 years since the appearance of the first edition of *Rose and Carless* and the present 18th edition remains as invaluable a textbook for our present students as the previous editions have been to several generations of their predecessors.

Sir Cecil Wakely has set out to bring this edition thoroughly up to date and he has accomplished the difficult task admirably. The many advances that have been made in all branches of surgery have been covered thoroughly and systematically. Many chapters have been completely rewritten and new chapters have been included. Thus there are now very useful sections on *Water and Salt Deficiency in Surgery, Biopsy in Surgery, The Pathogenesis of Infection, Chemotherapy, Disorders of the Blood, Blood Transfusion, Haemorrhage and Shock, The use of Physical Agencies in Surgery, Burns and their Treatment* and on *Plastic Surgery*.

Eighteen specialists have contributed to this edition and so ensured that the latest and most practical information in their specialities is made available to the reader. The chapter on *Burns* by Mr. Wallace merits special mention.

The sections on the treatment of thyroid disorders and the use of radio-iodine, thymectomy in myasthenia gravis, the surgery of the heart and the great vessels, pancreatectomy and the management of intestinal obstruction serve to illustrate the comprehensiveness of the work and the great trouble that has been taken by the Editor to bring the edition completely up to date. The chapter on the rectum and anus follows the teaching of the St. Mark's Hospital and the minor rectal conditions which are so important to the practitioner are dealt with in a thorough and lucid manner. The chapter on amputations, based on experiences during the recent war, is equally useful.

Numerous new illustrations, together with several coloured plates, have been incorporated and the text is well set out under large headings in heavy print.

It is an admirable text-book for the undergraduate, while the practitioner will not fail to find in it the answers to surgical problems which may arise in his practice. The new edition should indeed live up to the Editor's expectations of being 'a guide, philosopher and friend' to the student and as well as the practitioner.

THE NORMAL CHILD

The Normal Child. Some Problems of the First Three Years and Their Treatment. By Ronald S. Illingworth, M.D., F.R.C.P., D.P.H., D.C.H. (Pp. 342, with Tables. 30s.) England: J. & A. Churchill Limited.

Contents: *Section I. Feeding Problems.* 1. Breast Feeding or Artificial Feeding? 2. The Feeding Schedule. 3. Some Breast Feeding Problems. 4. Breast and Nipple Difficulties. 5. Insufficiency of Milk. 6. The Failure of Lactation. 7. Difficulties in the Breast-Fed Baby. 8. Difficulties in the Artificially-Fed Baby.

Section II. Physical Problems. 9. The Assessment of Physical Development. 10. The Skull. 11. The Teeth. 12. The Skin and Umbilicus. 13. The Breast and Genitals. 14. Some Miscellaneous Problems. 15. The Prevention of Infection.

Section III. Developmental Problems. 16. Is Prediction Possible?. 17. The Normal Course of Development. 18. General Factors Which Affect the Course of Development. 19. Retardation in Single Fields of Development and Factors Responsible. 20. Developmental Diagnosis.

Section IV. Behaviour Problems. 21. Relevant Features of the Psychological Development of the Child. 22. Parental Attitudes and Management. 23. Discipline and Punishment. 24. Some General Results of Unsatisfied Needs. 25. Anorexia and Other Eating Problems. 26. Sleep Problems. 27. Problems of Sphincter Control. 28. Crying, Temper Tantrums, Breath-Holding Attacks. 29. Body Manipulations. 30. Jealousy, Fears, Shyness. 31. Some Other Problems. References. Additional Reading Recommended. Index.

This book sets out to describe the problems other than disease commonly encountered in the normal child during the first 3 years of life.

The newly qualified practitioner, fully equipped with a knowledge of the diseases of childhood but more dimly aware of these frequent and often distressing problems, will welcome this book.

The author has not contributed any new or radical views in this field, but has performed a useful function in setting out clearly in one extremely readable book this much needed information.

DEVELOPMENT CHARTS

Blum-Fieldsteel Development Charts. By Lucille Hollander Blum and Nina D. Fieldsteel. (Each package contains 25 of each Chart and a Manual of Directions. $2.50 plus postage per package.) New York: World Book Company.

The activities listed on these charts represent a careful selection from those included in the Gesell Developmental Schedules, and their use presupposes a knowledge of these tests as set out in Gesell and Amatruda's *Developmental Diagnosis*. With the 2 charts depicting motor and functional behaviour comes a *Manual of Directions* clearly explaining the method of administering the tests and of recording and evaluating the results.

These charts provide an interesting and useful method of recording and correlating information on the development of

the child. Whether or not they would prove of practical value to the paediatrician or psychologist in the individual case will depend on the cost of the printed charts and the time taken to employ them. They should, however, be of great value where large numbers of children are being investigated for the purpose of obtaining comparative data. Use can also be made of these charts to portray changes observed during therapy. On the whole they may be regarded as an interesting and practical contribution to the field of child development.

CORRESPONDENCE

THYROID TREATMENT ; THE CONTROL OF EXPERIMENTS

To the Editor: Dr. Menof's letter in this *Journal* (4 April 1953) raises certain fundamental issues regarding the interpretation of experimental findings in therapeutics.

Bertrand Russell [1] described the scientific outlook as a refusal to regard our own desires, tastes and interests as affording a key to the understanding of the Universe. Yet therapeutics, the most experimental of all sciences, has remained the least affected by true scientific objectivity. Conceived in traditions of witchcraft, throughout centuries, subjective influences have confounded the true scientific appraisal of the value of drugs.

Dr. Menof states that the control of experiments is a vexed question. There cannot be any doubt about experimental control, which is an application of scientific method to render, as far as possible, a degree of objectivity to subjective phenomena. Unless such control is applied, our knowledge of drugs, instead of being well defined, becomes chaotic. The use of Vitamin E in cardiac pain is certainly a vexed question. The uncontrolled experiments relying on the subjective impressions of patients (Vogelsang *et al.*[2] Gram and Schmidt [3]) demonstrated markedly good results. Yet when Ringler *et al.*[4] published their findings in a carefully controlled experiment, Vitamin E was found to be of no benefit in such cases.

The very essence of control is to neutralize those natural effects which Dr. Menof describes; in regard to blood pressure-anxiety, mental rest, etc. In a *random* sample *both* groups would show changes which could be clearly assessed as significant or not according to environmental influences. The effect of the drug in question could likewise be measured and assessed according to well-known statistical principles. If there is any doubt in the result, the experiment could be replicated.

Unless the trial is well planned the reliability of the result will be in doubt. Dr. Menof is convinced that an individual showing a good response to a drug given alternatively with a placebo which had no effect, demonstrates that this drug is of value. Surely, in this type of experiment it is impossible to identify the psychological and environmental influences and separate them from the specific effect of the drug to be tested.

The problems involved in the planning of experiments are admirably elucidated by Bradford Hill [5] in the *British Medical Bulletin* in an issue covering the whole field of medico-metrics.

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S. B. Sachs.

P.O. Evaton,
Transvaal.
12 April 1953.

THE LATE DR. N. A. STUTTERHEIM

To the Editor: My contact with Dr. Stutterheim extended over 16 years and I have been unusually privileged in getting to know both his ophthalmological views and his unique personality. I followed with considerable interest the development of his ideas on the origin of bifoveal vision in involuntary convergence (as distinct from adduction). It may therefore be profitable to attempt to outline his theory and practice.

Dr. Stutterheim measured involuntary or reflex convergence by asking the patient to fix his gaze on a distant object, while a succession of prisms was passed in front on one eye until the patient saw 'double'. The range of prism strengths that could be endured without diplopia represented an estimate of the power of reflex convergence.

He found a great reduction in this range in patients complaining of eyestrain, e.g., instead of the normal range of 50 to 60 degrees of prism they might tolerate less than 10. Repeated sessions with the prism battery would improve a patient's range to normal and better, by facilitation of the existing reflex mechanism. The symptomatic results of his treatment were often dramatic, and minor refractive errors could then be ignored. Thus many of his patients—myself included—subsequently discarded spectacles, though his aim was never to 'strengthen the eyes'. During the war his views received great encouragement, since it was found that many pilots who remained persistently 'bad landers' notwithstanding the stringent visual tests required for admission, showed greatly improved flying performances after treatment for co-existent eyestrain.

In squint, Stutterheim held, the power of convergence is in abeyance, ocular deviation representing one method of escape from what would otherwise be intolerable diplopia. If the latent ability of convergence could be developed, the squinter would be cured, though certain complications of squinting, e.g., anatomical contracture and pseudo-fovea might remain and need separate treatment. His first step was to develop true bifoveal vision, using prisms to re-direct an image on to the fovea of the squinting eye. Once fusion had been achieved, the range of convergence was extended by varying the prism strength just as in the case of eyestrain. The rate of improvement might be hastened by surgical correction of the long-standing postural defect of the squinting eye, a fact that was frequently misunderstood, but Stutterheim asserted that he did not 'operate for squint', for the squint was already cured before the adjustment was made. Simple parallelization of optic axes without previous achievement of bifoveal vision he regarded as a purely cosmetic operation, but not the physiological restoration of normal function at which he aimed.

He once observed to me, somewhat wryly, that an ophthalmological qualification appeared to be the only real barrier to an understading of his ideas, for they were readily grasped by physiologists and general practitioners. To some of his colleagues however, he seemed to be a charlatan claiming to strengthen the eyes by exercises, while to others he was making an unnecessary fuss over what was already familiar and established practice, like that of putting a bird seen with one eye into a cage seen with the other. Stutterheim used synoptoscopes and similar devices, but insisted that involuntary convergence was different from superimposition of the images of unlike or separate objects.

My grasp of his ophthalmological views must necessarily be limited: it was as a man that he made the deepest impression on me. He had an intense love of nature, and derived constant delight in the wild life of this country. His home was open to many who could not hope to return his hospitality —as a young medical student, I was a regular visitor—and with a courtesy more characteristic of an earlier age, he could make each guest feel he was conferring the favour in being present. His humble, gentle, yet dignified spirit sprang from a deep and real Christian faith, and it was typical of the man that though he was ready to speak of his belief, it was only in the last year of my acquaintance that, in answer to direct questioning he disclosed the extent of his theological training. His faith was based on sounder foundations than 5 years of theology, and it was not in his nature to draw attention to any disparity that might exist between himself and others.

A. G. Oettlé.

S.A. Institute for Medical Research,
Johannesburg.
11 April 1953.

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The Medical Association of South Africa : Die Mediese Vereniging van Suid-Afrika

AGENCY DEPARTMENT : AGENTSKAP-AFDELING

JOHANNESBURG

Medical House, 5 Esselen Street. Telephone 44-9134-5, 44-0817
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(L V372) Transvaal hospital town. Locum for 6 months as from 1 June. Salary £80 p.m., plus garage account paid.
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DURBAN

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PRACTICES FOR SALE : PRAKTYKE TE KOOP

(PD13) Natal Lower South Coast practice, near Pondoland border, suitable for retired doctor. Area developing and large Police holiday camp in vicinity. Excellent climate and very good fishing. Premium required £400, includes good stock of drugs and dressings, instruments and dispensary furniture. House for sale £1,800, including stand of one-third morgen. Bond available. For immediate sale. Owner having taken a full-time appointment.
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(PD18) Natal Midlands. Excellent prospects in rapidly developing area. General mixed practice. Seller going overseas. Premium £1,500 includes surgery furniture, fittings, instruments. Total gross receipts for 1950, £2,691; 1951, £2,709; 1952, £2,573. Ideal climate and sporting facilities. For immediate sale.
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(136) Near Durban. Locum from about the last week in May, or for the month of June. £2 12s. 6d. per day, all found. Own car necessary.
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(1335) Transkei hospital town. Assistant from July 1953 with definite view to partnership. Commencing salary £60—£70 p.m. according to experience.
(1328) Western Province. From early June or as soon as possible thereafter for 2 months in partnership practice. £2 2s. per day, plus board and lodging, petrol and car allowance.
(1305) Westelike Provinsie hospitaa'dorp. Vanaf 16 Mei tot einde Junie. Salaris £2 12s. 6d. per dag, alles vry. Indien locum sy eie kar het, 9d. per myl kartoelaag.
(1354) Bolandse hospitaaldorp. Vanaf 20 Junie vir een maand, en weer vanaf 1 Desember vir een maand. Salaris £2 12s. 6d. plus 8d. per myl kartoelae. Reiskoste heen en terug.

KOOP VAN VENNOOTSKAPSAANDEEL

(1110) 'n Geneesheer met 'n heel paar jaar ondervinding in sy eie algemene praktyk stel belang in 'n vennootskap verkieslik in 'n hospitaaldorp, met 'n kollega wat taamlik snywerk doen. 'n Assistentskap met die oog op latere vennootskap sal ook oorweeg word.

Provincial Administration of the Cape of Good Hope

HOSPITALS DEPARTMENT

HOSPITAL BOARD SERVICE: VACANCIES

1. Applications are invited from registered medical practitioners for appointment to the two posts of Medical Superintendent for the groups of hospitals stated hereunder:

Institution	*Post*	*Emoluments*	*Closing date*	*Applications to be addressed to:*
Group I				
Victoria Hospital, Wynberg *Eaton and McGregor Convalescent Homes* *Princess Alice Home of Recovery* *Lady Michaelis Orthopaedic Home*	Medical practitioner, Grade C, (Medical Superintendent).	£1,000 × 50–1,200 p.a.	29.5.53	The Director of Hospital Services, P.O. Box 2060, Cape Town.
Group II				
Woodstock Hospital *Rondebosch & Mowbray Hospital* *Mowbray Maternity Hospital* *Peninsula Maternity Hospital*	Medical practitioner, Grade C, (Medical Superintendent).	£1,000 × 50–1,200 p.a.	29.5.53	The Director of Hospital Services, P.O. Box 2060, Cape Town.

2. Applications are invited from registered medical practitioners for appointment to the following vacant posts:

Group III				
Eaton and McGregor Convalescent Homes	Medical practitioner, (part-time).	£180 p.a. (fixed).	29.5.53	The Director of Hospital Services, P.O. Box 2060, Cape Town.
Kimberley Hospital, Kimberley	Medical practitioner, Grade E, (Radiologist).	£1,600 p.a. (fixed).	29.5.53	The Director of Hospital Services, P.O. Box 2060, Cape Town.

3. Conditions of service are prescribed in terms of Hospital Board Service Ordinance No. 19 of 1941, as amended, and the regulations framed thereunder.

4. In addition to the scale of salary indicated a cost of-living allowance at rates prescribed from time to time by the Administrator is payable to whole-time officials and employees.

5. The successful candidates for the two posts of Medical Superintendent will be required to occupy, free of charge, an unfurnished house or quarters provided at the institution or alternatively, if a house or quarters are not available to occupy a house approved by the Department in respect of which the Department will contribute an amount of not exceeding £180 per annum towards the rental.

6. The successful candidates, if not already in the Hospital Board Service, will be required to submit satisfactory birth and health certificates.

7. Application must be made on the prescribed form (Staff 23) which is obtainable from the Director of Hospital Services, P.O. Box 2060, Cape Town, or from the Branch Representative of the Hospitals Department, P.O. Box 1487, Cape Town, or from the Medical Superintendent of any Provincial Hospital or Secretary of any School Board in the Cape Province.

8. Candidates must state the earliest date on which they can assume duty. (A562967)

Provinsiale Administrasie van die Kaap die Goeie Hoop

HOSPITAALDEPARTEMENT

HOSPITAALRAADSDIENS: VAKATURES

1. Aansoeke word ingewag van geregistreerde geneeshere vir aanstelling tot die twee poste van Mediese Superintendent vir die Hospitaal-groepe hieronder gemeld:

Inrigting	*Pos*	*Emolumente*	*Sluitingsdatum*	*Aansoeke moet gerig word aan:*
Groep I				
Victoriahospitaal, Wynberg *Eaton- en McGregorherstellingstehuise* *Princess Alice-herstellingstehuis* *Ortopediese Tehuis Lady Michaelis*	Geneesheer, Graad C, (Mediese Superintendent).	£1,000 × 50–1,200 p.j.	29.5.53	Die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad.
Groep II				
Woodstockhospitaal *Rondeboschen Mowbrayhospitaal* *Mowbraykraamhospitaal* *Skiereilandse Kraamhospitaal*	Geneesheer, Graad C, (Mediese Superintendent).	£1,000 × 50–1,200 p.j.	29.5.53	Die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad.

2. Aansoeke word ingewag van geregistreerde geneeshere vir aanstelling tot die volgende vakante poste:

Groep III				
Eaton- en McGregorherstellingstehuise	Geneesheer (deeltyds).	£180 p.j. (vasgestel).	29.5.53	Die Direkteur van hospitaaldienste, Posbus 2060, Kaapstad.
Kimberleyhospitaal, Kimberley	Geneesheer, Graad E, (Radioloog).	£1,600 p.j. (vasgestel).	29.5.53	Die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad.

3. Die diensvoorwaardes word voorgeskryf ingevolge die Ordonnansie op Hospitaalraadsdiens nr. 19 van 1941, soos gewysig, en die regulasies wat daarkragtens opgestel is.

4. Benewens die salarisskaal soos aangedui is 'n lewenskostetoelae betaalbaar aan voltydse beamptes en werknemers teen bedrae wat van tyd tot tyd deur die Administrateur vasgestel word.

5. Van die geslaagde kandidate vir die twee poste van Mediese Superintendent sal dit vereis word om 'n ongemeubileerde huis of kwartiere wat by die hospitaal verskaf word gratis te bewoon, of as 'n huis of kwartiere nie beskikbaar is nie, 'n huis te bewoon wat deur die Departement goedgekeur is ten opsigte waarvan die Departement 'n bedrag van hoogstens £180 per jaar tot die huur sal bydra.

6. Die geslaagde kandidate, indien nie reeds in die Hospitaalraadsdiens nie, moet bevredigende geboorte- en gesondheidsertifikate indien.

7. Aansoek moet gedoen word op die voorgeskrewe vorm (Staf 23) wat verkrygbaar is by die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, of by die Takverteenwoordiger van die Hospitaaldepartement, Posbus 1487, Kaapstad, of by die Mediese Superintendent van enige provinsiale hospitaal of by die Sekretaris van enige Skoolraad in die Kaapprovinsie.

8. Applikante moet die vroegste datum meld waarop hulle diens kan aanvaar. (A562967)

Transvaalse Provinsiale Administrasie

VAKATURES BY PUBLIEKE HOSPITALE

Aansoeke word ingewag van kandidate met geskikte kwalifikasies vir die onderstaande poste by Publieke Hospitale in die Transvaal.

Aansoeke moet gerig word aan die Geneeskundige Superintendent of Verantwoordelike Geneesheer van die betrokke Hospitaal en moet volle besonderhede bevat aangaande die ouderdom, professionele, akademiese en taalkwalifikasies, ondervinding en huwelikstaat van die applikant en moet voorts 'n aanduiding bevat van die vroegste datum waarop diens aanvaar kan word:

Lewenskostetoelae tans betaalbaar aan voltydse werknemers:

Salaris	*Lewenskostetoelae*	
	Getroud	*Ongetroud*
Oor £350	£320 p.j.	£100 p.j.

Van persone wat aangestel word, sal verwag word om bevredigende sertifikate in te dien, asook om hulle te onderwerp aan 'n geneeskundige ondersoek by die betrokke hospitaal.

Aansoek vorms is verkrygbaar van enige Transvaalse Publieke Hospitaal of die Provinsiale Sekretaris, Afdeling Hospitaaldienste, Posbus 2060, Pretoria.

Benewens jaarlikse salaris en lewenskostetoelae ontvang voltydse werknemers spoorwegkonsessie en word verlof toegestaan ooreenkomstig die hospitaal verlofregulasies.

Die sluitingsdatum van aansoeke vir die poste is 18 Mei 1953.

Hospitaal	*Vakature*	*Emolument*	*Opmerkings*
Germiston	Ongevalle beampte (1)	£620, 780, 820, 860	Geregistreerde mediese praktisyn. Moet vir ten minste 2 jaar gekwalifiseerd wees.
Pretoria	Kliniese Assistent (Departement van Kindergeneeskunde) (1)	£620, 780, 820, 860	Geregistreerde mediese praktisyn. Suksesvolle applikante moet bereid wees om op 1 Augustus 1953, diens te aanvaar.

(40750)

Public Service Commission

VACANCIES IN THE PUBLIC SERVICE

1. The attention of medical practitioners, registered with the South African Medical and Dental Council, is drawn to an advertisement appearing in the *Government* and *Provincial Gazettes* of this week, inviting applications for the undermentioned posts:

Post	*Department*	*Salary Scale*
Medical Officer	Health (Nelspoort Sanatorium)	£900 × 50—1,150
Medical Officer (on contract for 2 years)	Health (Stellenbosch)	£900 × 50—1,150
District Surgeon, Grade III	Health (Pietersburg and Laersdrift)	£900 × 50—1,150

2. In addition to salary a cost-of-living allowance at the rate of £320 per annum (married) and £100 per annum (single) is payable at present.

3. It is emphasized that full and detailed particulars of qualifications and previous experience must be furnished but original certificates and testimonials should not be submitted. Application forms Z.83 and P.S.C. 8(a) are obtainable from the Secretary, Public Service Commission, Pretoria, to whom filled-in forms must be addressed.

4. The closing date for the receipt of applications is 30 May 1953.

(40799)

Provincial Administration of the Cape of Good Hope

HOSPITALS DEPARTMENT

SHARLEY CRIBB NURSING COLLEGE, PORT ELIZABETH: LECTURES TO STUDENT NURSES

Applications are invited from registered medical practitioners to lecture student nurses at the Sharley Cribb Nursing College, Port Elizabeth, in the following subjects for the period 1 June 1953, to 30 November 1954.

Surgical nursing (English lectures), 40 lectures per course: 3 courses per annum.

Lectures are to be given between the hours 8 a.m. and 3 p.m. daily, each lecture to be of one hour's duration.

Lecturers will be remunerated at the rate of £1 1s. per lecture.

Further particulars will be obtained from the Principal, Sharley Cribb Nursing College, Park Drive, Port Elizabeth.

Application must be made on the prescribed form Staff 23 which is obtainable from the Director of Hospital Services, P.O. Box 2060, Cape Town, or from the Branch Representative of the Hospitals Department, P.O. Box 1487, Cape Town, or from the Medical Superintendent of any Provincial Hospital or Secretary of any School Board in the Cape Province.

Applications must be addressed to the Director of Hospital Services, P.O. Box 2060, Cape Town, and must reach him not later than 18 May 1953.

(A562957)

Provinsiale Administrasie van die Kaap die Goeie Hoop

HOSPITAALDEPARTEMENT

SHARLEY CRIBB-VERPLEGINGSKOLLEGE, PORT ELIZABETH: LESINGS VIR LEERLINGVERPLEEGSTERS

Aansoeke word ingewag van geregistreerde geneeshere om lesings aan leerlingverpleegsters aan die Sharley Cribb-verplegingskollege, Port Elizabeth, te gee oor die volgende onderwerpe vir 'n tydperk 1 Junie 1953 tot 30 November 1954.

Chirurgiese Verpleging (Engelse lesings), 40 lesings per kurses: 3 kursusse per jaar.

Lesings moet gegee word tussen die ure 8 vm. en 3 nm. daagliks. Elke lesing sal een uur duur.

Lektore sal besoldig word teen £1 1s. per lesing.

Nadere besonderhede is verkrygbaar by die Prinsipale, Sharley Cribb-verplegingskollege, Park Drive, Port Elizabeth.

Aansoek moet gedoen word op die voorgeskrewe vorm (Staf 23) wat verkrygbaar is by die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, of by die Takverteenwoordiger van die Hospitaaldepartement, Posbus 1487, Kaapstad, of by die Mediese Superintendent van enige provinsiale hospitaal of by die Sekretaris van enige Skoolraad in die Kaapprovinsie.

Aansoeke moet aan die Direkteur van Hospitaaldienste, Posbus 2060, Kaapstad, gerig word en moet hom nie later as 18 Mei 1953 bereik nie.

(A562957)

Witwatersrand Jewish Aged Home

Applications are invited from general practitioners in the Johannesburg area for the position of honorary visiting doctor to the Witwatersrand Jewish Aged Home. There are 3 vacancies to be filled. A knowledge of Yiddish is desirable. Applications to be addressed to Chairman of the Medical Committee, 704 Medical Centre, Johannesburg.

City of Johannesburg

VACANCY

Applications are invited for the following vacant position in the City Health Department:

Consultant Thoracic Surgeon: £218 per annum, including cost-of-living allowance, plus locomotion allowance of 6½d. per mile.

Applicants must be Specialist Thoracic Surgeons, registered to practice as such in South Africa. The appointee will be called upon to render consultant services as required, but mainly at the Tuberculosis Clinics and the hospital maintained by the Council.

The time involved will be approximately 4 hours per week.

No transport allowance will be paid for journeys made to places within a 3-mile radius of the incumbent's consulting rooms.

Personal canvassing for appointment in the gift of the Council is strictly prohibited. Proof thereof shall disqualify a candidate for appointment.

Applications in the candidates' own handwriting on special forms to be obtained from the Central Staff Office, Room 223, Municipal Offices, must be placed in the box in Room 223, Municipal Offices, or posted so as reach the Town Clerk, not later than 4 p.m., 14 days after publication of the *South African Medical Journal*.

D. Ross-Blaine
Acting Town Clerk
(A2238/106)

17 April 1953
(145)

Bridgman Memorial Hospital, Johannesburg

HOUSE SURGEONS IN OBSTETRICS

Applications are invited from medical practitioners for 3 posts of house surgeon in obstetrics at the above non-European Maternity Hospital for the period 1 August 1953 to 31 January 1954 inclusive. Successful applicants may be required to lecture to pupil midwives.

Salary £240 per annum plus married or single cost-of-living allowance, board, lodging and laundry.

Closing date for applications: 18 May 1953.

Applications with a complete list of previous experience should be sent to the Superintendent, Bridgman Memorial Hospital, High Street, Mayfair, Johannesburg.

Practice for Sale

Northern Natal. An unopposed dispensing practice in a wealthy farming district. Annual gross income £3,200, including D.S. appointment and big Native cash income. Practice, drugs, instruments and a Jeep (useful in wet weather) for £2,500. A 9 roomed house, 7 roomed surgery, 3 garages, Native quarters, etc. all for £3,500. Total £6,000. Owner selling for health reasons. Write 'A. Q. E.', P.O. Box 643, Cape Town.

Salford Royal Hospital

SALFORD HOSPITAL MANAGEMENT COMMITTEE

Applications are invited for the post of house surgeon, Ear, Nose and Throat and Neurosurgical Departments. The appointment is for 6–8 months. Vacant immediately. The post, which is resident, is in the salary range £350—£450 per annum. Applications, with copies of 3 testimonials, should be sent to the Secretary, Salford Royal Hospital, Salford, Lancs., England.

Port Elizabeth Tramway Employees' Sick Fund

WANTED: MEDICAL OFFICER

Applications are invited from registered medical practitioners for the post of Medical Officer to the Port Elizabeth Tramway Employees' Sick Fund.

Applications must be lodged with the undersigned (from whom all information may be obtained) not later than 12 noon on Saturday, 16 May 1953.

Secretary.
P.O. Box 1069
Port Elizabeth

(This appointment has the approval of the Medical Association.—*Assistant Secretary.*)

Suite To Let

Professional suite available at Mayo Centre, corner Fortescue and Raleigh Streets, Yeoville, Johannesburg, for a physiotherapist. All other professional rooms are occupied by dental and general medical practitioners. This area is very thickly populated and a physiotherapist would meet the urgent need in the district. Apply by letter, or telephone the Rents Manager (I. Seeff personal) S. Seeff and Company Ltd., 134 Fox Street, Johannesburg. Telephone: 23-9715.

Partnership Wanted

Fully bilingual, gentile doctor with capital, wishes to purchase a partnership in a well-established practice, or a practice in one of the larger country towns in the Union, preferably in the Western Cape or at the coast. This doctor has been conducting a large practice for the past 8 years and will only consider a first-class proposition. Write 'A. Q. R.', P.O. Box 643, Cape Town.

Partnership Offered

Partnership offered to anaesthetist specialist in flourishing solus anaesthetic practice. Large centre. Reasonable premium and terms offered. Reply, stating age, experience, marital status and date when free to commence, to 'A. Q. H.', P.O. Box 643, Cape Town.

Practice for Sale

Flourishing dispensing mixed practice in Cape Peninsula. Available with above is a modern comfortable home in Southern Suburbs; also with surgery and waiting room. Apply 'A. Q. L.', P.O. Box 643, Cape Town.

Wanted

One Physiotherapist to work full-time at Rehabilitation Centre in Gwelo, Southern Rhodesia. Salary £40 per month. Accommodation provided at site at £9 per month. Reply: Secretary, Poliomyelitis Rehabilitation Centre, P.O. Box 484, Gwelo, Southern Rhodesia.

Assistant Required

Professional assistant required, full time or part-time, male or female. Industrial practice, Cape Town. Duties to commence on 1 July 1953. Write 'A. Q. G.', P.O. Box 643, Cape Town.

Practice for Sale

Non-European dispensing practice. Just outside Johannesburg. Three surgeries serving 3 locations. Telephone Johannesburg 44-7057 between 6–7 p.m. or write 'A. Q. P.', P.O. Box 643, Cape Town.

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MEDICAL HOUSE, 35 Wale Street, Cape Town. P.O. Box 643. Telephone 2-6177. Telegrams: 'Medical'